COLUMBIA LIBRARIES OFFSITE
HEALTH SCIENCES STANDARD
HX00015881

MEDICAL BOOKS
69E.59th St., N.Y.

RC 341 Sp3

Columbia University
in the City of New York
College of Physicians and Surgeons

Reference Library

Digitized by the Internet Archive
in 2010 with funding from
Open Knowledge Commons

http://www.archive.org/details/manualofnervousd00spea

A MANUAL

of

NERVOUS DISEASES

By

IRVING J. SPEAR, M. D.

Professor of Neurology at the University of Maryland, Baltimore

WITH 172 ILLUSTRATIONS

PHILADELPHIA AND LONDON

W. B. SAUNDERS COMPANY

1916

Med
17-26650

Copyright, 1916, by W. B. Saunders Company

RC341
Sp3

PRINTED IN AMERICA

PRESS OF
W. B. SAUNDERS COMPANY
PHILADELPHIA

RESPECTFULLY DEDICATED TO MY
FRIEND AND COLLEAGUE

PROFESSOR RANDOLPH WINSLOW, A. M., M. D., LL. D.

PREFACE

THE preparation of this volume for the present and for the future general practitioner has been undertaken in the hope that I have been able to embody in a book of moderate size the facts necessary for a proper understanding of the anatomy, the physiology, and the diseases of the nervous system.

In my experience as teacher and consultant I have found that the student of medicine, as well as the general practitioner, regards the study of diseases of the nervous system as particularly difficult. This false impression I attribute to a lack of a proper understanding of the anatomy and physiology of the nervous system, and of the correct methods of examining nervous patients. This lack of knowledge may be due in some measure to the fact that there is not at present available a moderately brief exposition limited to a description of the practical anatomy and physiology of the nervous system. Such a description, to be useful, must be neither too brief nor too voluminous, but should contain those facts and theories essential to a clear understanding of the mechanism of organic nervous diseases, and it is this deficiency that I have attempted to supply in the first part of this volume.

No effort has been made completely to cover the subject of neurology, but I have endeavored to treat it with sufficient fulness to make a comprehension of the salient features of organic nervous diseases easily possible. This I hope further to have facilitated by the aid of illustrations and diagrams, some of which are original, whereas others have been taken from the various text-books on anatomy, physiology, neurology, and kindred subjects, to which due credit has been given.

The method of examination laid down in this volume appears to me to be that best suited to the use of the general practitioner

and the student, since it requires no special apparatus beyond a galvanic and a faradic battery, demands very little special training, and is easily remembered.

The second part of this volume is devoted to a consideration of the diseases of the nervous system. Of these, the first to be considered are those that are readily understood; for example, the diseases of the peripheral nerves, of the muscles, of the spinal cord, etc. These are followed by the diseases having no apparent pathologic basis, the last to be considered being those diseases due to perversion of the functions of the ductless glands and the sympathetic system. Thus by considering first the simpler diseases, the way is gradually prepared for placing within the grasp of the student the more difficult subjects.

I have endeavored to describe each disease as clearly and briefly as possible, and to devote to each subject the amount of space that, in my judgment, its importance demanded.

Special attention has been given to differential diagnosis and to treatment, and I have, in the main, confined myself to a consideration of the most recent accepted facts, avoiding, so far as possible, lengthy discussions and theories.

My thanks are due to Mr. Chandlee for his painstaking care in the making of many of the photographs, to my medical friends for kind assistance, and to my stenographer, Miss Gant, for her untiring and intelligent interest.

IRVING J. SPEAR.

BALTIMORE, MD.,
November, 1916.

CONTENTS

ANATOMY AND PHYSIOLOGY OF THE NERVOUS SYSTEM

PAGE

Macroscopic Examination of the Nervous System 19

The Neuron 23

Anatomy and Physiology of a Peripheral Nerve 29

Anatomy and Physiology of the Spinal Cord 32

Structure of the Spinal Cord 40
Blood-supply of the Spinal Cord 50
Physiology of the Spinal Cord 53

Anatomy and Physiology of the Medulla Oblongata, Pons, and Crura 58

Structure of the Medulla, Pons, and Cerebral Peduncles 66

The Cerebellum 73

The Cerebrum 81

The Under Aspect of the Cerebrum 90
Interior of the Cerebrum 93
Blood-supply of the Cranial Contents 109
Functions of the White Substance of the Cerebrum 114
Function of the Cerebral Cortex 116

Anatomy and Physiology of the Cranial Nerves 123

The Sympathetic Nervous System 136

EXAMINATION OF THE PATIENT

History and Physical Examination 142

Physical Examination 145
Examination of the Nervous System 147

Reflexes 152

Sensory Functions 162

Electric Examination 167

Examination of the Cranial Nerves 172

Spinal Puncture 176

Craniocerebral Topography 178

DISEASES OF THE PERIPHERAL NERVES

PAGE

Special Forms of Traumatic and Inflammatory Diseases of the Spinal Nerves ... 186
Occupation Pareses ... 206
Multiple Neuritis (Peripheral Neuritis) ... 206
Special Forms of Multiple Neuritis ... 213
Herpes Zoster ... 222
Tumors of the Nerves ... 225
Neuralgia ... 226
Neuralgia of the Individual Nerves ... 230

DISEASES OF THE MUSCULAR SYSTEM

Progressive Muscular Dystrophy ... 239
Polymyositis ... 244
Ischemic Paralysis (Volkmann's Paralysis) ... 246
Thomsen's Disease (Myotonia Congenita) ... 247
Periodic Paralysis ... 249
Diseases of the Cranial Nerves ... 250
Diseases of the First or Olfactory Nerve ... 250
Disorders of the Second or Optic Nerve ... 251
Diseases of the Fifth Nerve ... 258
Diseases of the Seventh or Facial Nerve ... 259
Disorders of the Eighth or Auditory Nerve ... 263
Diseases of the Ninth or Glossopharyngeal Nerve ... 264
Diseases of the Tenth, Pneumogastric or Vagus Nerve ... 265
Diseases of the Eleventh or Spinal Accessory Nerve ... 267
Diseases of the Twelfth or Hypoglossal Nerve ... 267
Diseases of the Crura, Pons, and Medulla ... 268
Progressive Bulbar Paralysis ... 274

DISEASES OF THE SPINAL CORD

Acute Anterior Poliomyelitis ... 278
Subacute and Chronic Anterior Poliomyelitis (Progressive Spinal Muscular Atrophy) ... 286
Amyotrophic Lateral Sclerosis (Charcot's Disease) ... 289
Syringomyelia and Spinal Gliosis ... 292
Lateral Sclerosis ... 295
Ataxic Paraplegia ... 296
Friedreich's Disease (Hereditary Ataxia) ... 298
Locomotor Ataxia (Tabes Dorsalis) ... 303

PAGE
Pressure on the Spinal Cord 319
Fracture and Dislocation of the Spinal Column 325
Tumors of the Spinal Cord 331
Spinal Meningitis 339
Chronic Meningitis 341
Hypertrophic Pachymeningitis 342
Myelitis 344
Hematomyelia 349
Myelomalacia 353
Senile Paraplegia 355
Landry's Paralysis (Acute Ascending Paralysis) 357
Congenital Spinal Malformation (Spina Bifida; Rachischisis) .. 357

DISEASES OF THE BRAIN

Pachymeningitis 359
Pachymeningitis Externa 359
Pachymeningitis Interna 359
Meningeal Hemorrhage 361
Meningitis 364
Septic or Secondary Meningitis 365
Epidemic Cerebrospinal Meningitis 368
Tubercular Meningitis 372
Syphilitic Meningitis 375
Serous Meningitis—Meningismus 378
Cerebral Anemia 380
Cerebral Hyperemia 382
Rupture or Occlusion of the Cerebral Arteries 383
Thrombosis of the Cerebral Sinuses and Veins 403
Acute Non-suppurative Hemorrhagic Encephalitis 408
Infantile Cerebral Paralysis 412
Hydrocephalus 422
Abscess of the Brain 426
Cerebral Tumors 433
Paralytic Dementia (General Paresis; General Paralysis of the Insane) 449

DISEASES OF THE BRAIN AND SPINAL CORD

Syphilis of the Nervous System 459
Multiple Sclerosis (Disseminated Sclerosis) 465
Caisson Disease 469

DISEASES OF THE NERVOUS SYSTEM WITHOUT PATHOLOGIC FINDINGS

PAGE
Hysteria 471
Neurasthenia, or Nervous Exhaustion 493
Psychasthenia 511
Anxiety Neurosis 517
Occupation Neurosis—Occupation Spasms 518

NEUROSIS CHARACTERIZED BY SPASMODIC MUSCLE CONTRACTIONS

Facial Spasm 522
Spasms of the Muscles of Mastication 526
Spasmodic Torticollis 526
Spasmodic Affections of the Muscles of the Trunk and Extremities 530
Tic (Habit Spasm) 530
Chorea 533
Chorea Minor; Sydenham's Chorea or St. Vitus' Dance and Chorea Insaniens 534
Huntington's Chorea (Chronic Progressive Hereditary Chorea) ... 543
Chronic Progressive Chorea of Adults and Senile Chorea 545
Chorea Accompanying Organic Brain Lesions 547
Congenital Chorea 547
Epidemic Chorea 548
Electric Chorea 549
Paralysis Agitans (Shaking Palsy; Parkinson's Disease) 550
Epilepsy (Falling Sickness) 557
Myoclonus Epilepsy of Unverricht 572
Paramyoclonus Multiplex 573

DISEASES DUE TO PERVERSION OF SECRETION OF THE DUCTLESS GLANDS

Exophthalmic Goiter (Basedow's or Graves' Disease) 575
Myxedema—Cretinism 583
Tetany 589
Myasthenia Gravis 594
Disorders of the Pituitary Body 597
Gigantism 603
Acromegaly 605
Infantilism 611
Disorders of the Pineal Gland 615

DISEASES DUE TO DISTURBANCES OF THE VASOMOTOR SYSTEM

PAGE
Acroparesthesia 619
Migraine (Hemicrania) 620
Erythromelalgia 628
Raynaud's Disease (Symmetric Gangrene) 629
Angioneurotic Edema (Acute Circumscribed Edema) 636

TROPHONEUROSES

Progressive Facial Hemiatrophy 638
Scleroderma 640

UNCLASSIFIED DISORDERS

Achondroplasia 642
Tetanus 645

Index 651

MANUAL OF NERVOUS DISEASES

ANATOMY AND PHYSIOLOGY OF THE NERVOUS SYSTEM

Nervous tissue differs from other tissue in that, after a certain period, cells do not increase in number and never reproduce themselves. Their functional capacity and their durability are dependent upon the inherent and vital energy, the condition of the circumambient medium, and their capacity for the storage of energy.

The nervous system is composed of structures situated within the cranial cavity and in projections from this cavity; the spinal canal, from which projections reach every portion of the body, be it ever so small or remote; a series of structures with their projections situated in front of and on both sides of the spinal column, and connected by special forms of nerve tissue to the structures within the spinal canal and involuntary structures of the body. For descriptive purposes these portions are considered under the heads of the brain, which comprised that portion of the nervous system situated within the cranial cavity; the cranial nerves, consisting of those structures connected with the brain, and emerging from various openings in the skull; the spinal cord, that portion of the nervous system situated within the spinal canal; the peripheral nerves, those structures that convey impulses to and from the spinal cord; and the sympathetic or autonomic system, consisting of collections of cells and fibers situated on each side of and in front of the spinal column, and connected with the spinal cord by the rami communicantes, and

which communicate with and are distributed to every part of the body that contains smooth muscles, especially the blood-vessels, stomach, intestines, respiratory tract, urinary tract, salivary and sweat-glands, the glands of the gastro-intestinal tract, heart, esophagus, etc. (Fig. 1).

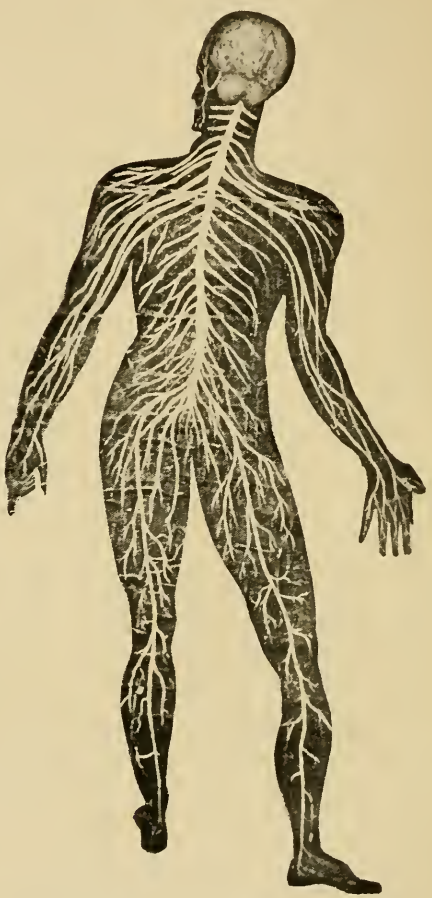


Fig. 1.—Brain, spinal cord, and peripheral nerves.

Before taking up the study of the structure or function of the separate parts of the nervous system, it is advisable to consider first its function as an entity. The nervous system receives stimuli from within and without the body. This constitutes the function of—(1) Irritability, or capacity for stimulation. It conducts stimuli to and from the nervous centers—(2) conductivity. It

reacts to stimuli either consciously or unconsciously—(3) conscious or automatic response. It regulates the distribution and force of the response to stimuli; that is, (4) regulation or government. It becomes impressed by stimuli—(5) Impressibility or apprehension. Finally it retains, or has the power of retaining and storing up, the memories of various stimulations—(6) memory.

These various functions are not subserved indiscriminately by all portions of the nervous system, certain structures being better adapted to carry on some functions, whereas others are capable of performing certain activities to better advantage. It is from a study of the perversion of these functions, from embryologic development, and from physiologic experimentation that we have been able to discover the parts played by many portions of the nervous system.

MACROSCOPIC EXAMINATION OF THE NERVOUS SYSTEM

Upon opening the cranial cavity we see a large mass, weighing in the adult about 1300 grams. This is the brain and its projections. The brain is covered by the *dura mater*, which is a dense, white membrane that is adherent to the inner surface of the skull. This membrane has several projections—an anteroposterior vertical one, called the falx cerebri, situated between two hemispheres attached anteriorly to the crista galli, and posteriorly and inferiorly to the tentorium cerebelli; a transverse process, called the tentorium cerebelli, which roofs the posterior cranial fossa and separates the under surface of the posterior cerebral lobes from the cerebellum (Fig. 2).

The dura mater extends down into the spinal canal, where it is separated from the surface of the bones by a layer of fat and venous plexuses. The dura sends tubular prolongations around the spinal nerves in their outward course, these prolongations being attached to the bones in the intervertebral foramina.

The function of the dura consists in helping to nourish the cranial bones to which it is adherent, to support the venous plexuses which it contains, to sustain the blood-vessels, and to protect and support the nervous elements which it covers (Fig. 3).

The important venous sinuses which the dura contains are the *longitudinal sinus*, which runs from before backward, and is formed at the superior junction of the falx cerebri with the dura; two *lateral sinuses*, which are situated at the posterior junction of the dura and tentorium cerebelli, in the attached margin of the latter; the *straight sinus*, formed at the junction of the falx cerebri

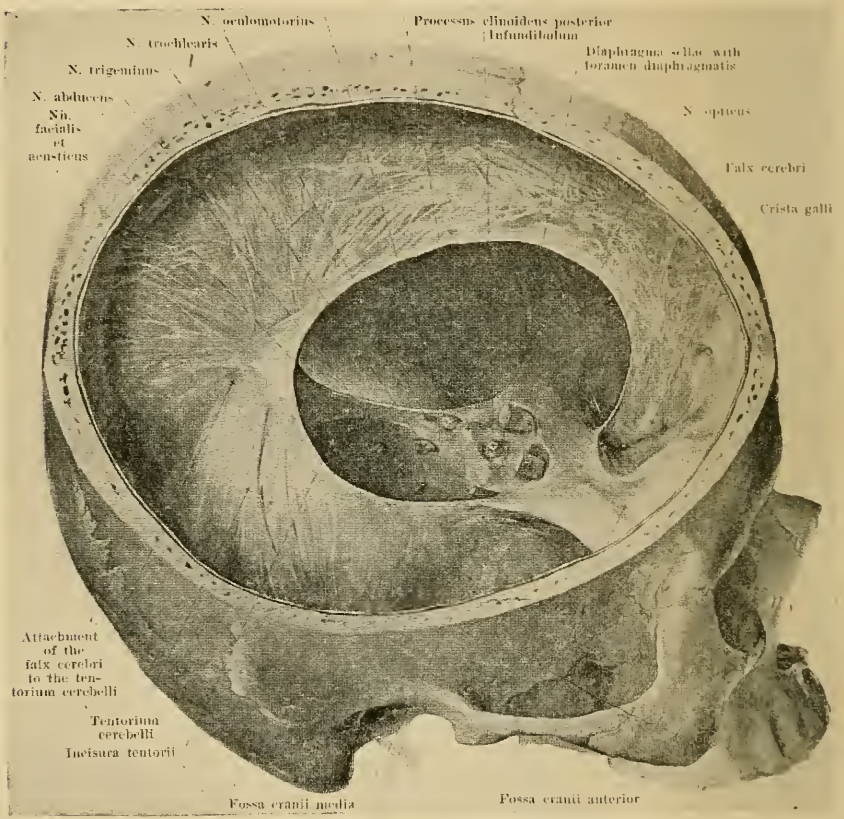


Fig. 2.—Dura mater (Spalteholz).

with the tentorium cerebelli; the *occipital sinus*, situated at the posterior attached margin of the falx cerebelli; the *inferior longitudinal sinus*, contained in the posterior part of the free margin of falx cerebri. The *torcular herophili* is situated at the junction of the superior longitudinal, lateral occipital, and the straight sinuses. In addition to these there are other sinuses situated in

the dura at the base of the brain. These are the cavernous, the circular, the transverse, and the superior and inferior petrosal sinuses.

Upon removing the dura we see, directly covering the brain, a very thin, transparent membrane; this is the *pia arachnoid*. The arachnoid is a delicate membrane that envelops the brain, lying between the pia mater and the dura mater, and being separated from the latter by the subdural space. It is separated from direct contact with the cerebral substance by the pia mater. It

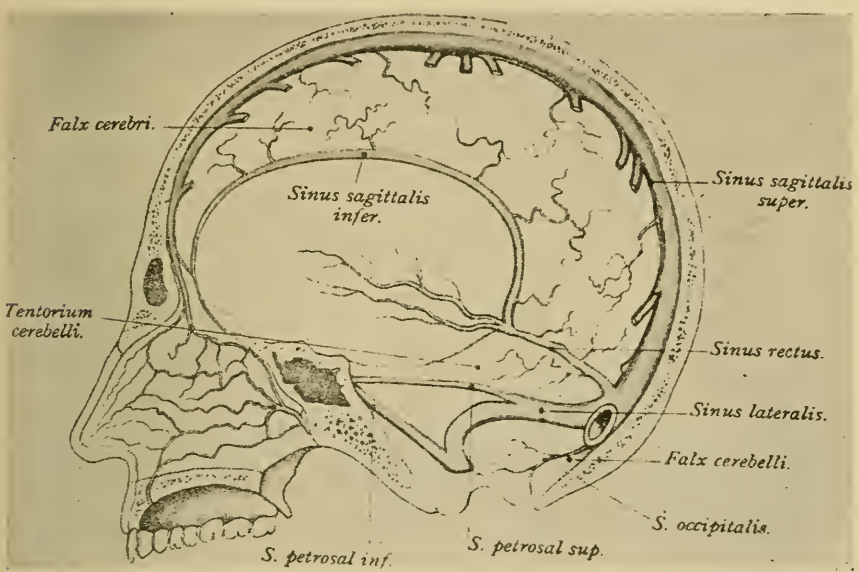


Fig. 3.—Venous sinuses (Whitaker, "Anatomy of the Brain and Spinal Cord").

passes over the convolutions without dipping down into the sulci between them. The *subarachnoid space* is the interval between the arachnoid and the pia mater, and it is this space that contains the cerebrospinal fluid. The subarachnoid spaces communicate with the general ventricular cavity of the brain by means of an opening in the inferior boundary of the fourth ventricle (Fig. 4).

The *pia mater* is a vascular membrane, and derives its blood supply from the internal carotids and vertebral arteries. It consists of a minute plexus of blood-vessels, bound together by an

extremely fine areolar tissue. The pia invests the entire surface of the brain, dipping down between the convolutions, and is prolonged into the interior, forming the velum interpositum and choroid plexuses of the fourth ventricle. Upon the surfaces of the hemispheres, where it covers the gray matter of the convolutions, it is very vascular, giving off from its inner surface a multitude of minute vessels that extend perpendicularly for some distance into the cerebral substance. At the base of the brain, in

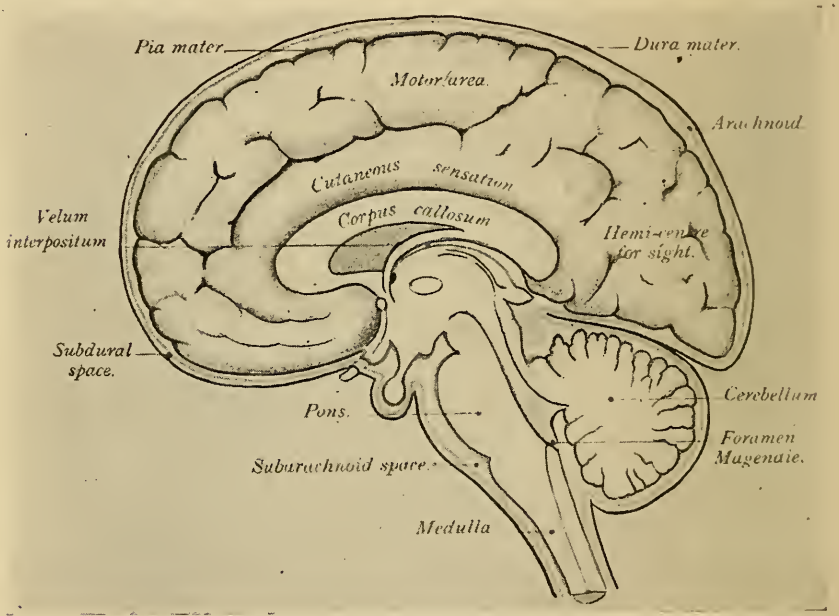


Fig. 4.—Arachnoid and pia mater (Whitaker, "Anatomy of the Brain and Spinal Cord").

the region of the anterior and posterior perforated spaces, a number of long straight vessels are given off that pass through the white matter to reach the gray substance in the interior. On the cerebellum the membrane is of more delicate texture, and the vessels coming from its inner surface are shorter. Upon the crura cerebri and pons Varolii the character of the pia undergoes a complete change, presenting here a dense fibrous structure marked only by slight traces of vascularity.

The cerebrospinal fluid fills the subarachnoid space and ven-

tricles. It is a clear, limpid fluid, having a slightly salty taste and a feeble alkaline reaction. According to Lassaigne, the cerebrospinal fluid consists of 98.5 parts of water, the remaining 1.5 per cent. being made up of solids, animal and saline. The fluid varies in quantity, being most abundant in old persons, and is quickly reproduced when lost.

The *brain* is a large oval mass, the surface of which contains many indentations or fissures. These include a deep antero-posterior depression, known as the great longitudinal fissure, and another, situated posteriorly, and called the transverse fissure. In addition to these there are many other depressions that divide the brain into smaller parts.

The *spinal cord* is a direct continuation of the brain into the spinal canal. Like the brain, it is covered by the dura and the pia arachnoid. During fetal life the spinal cord extends to the lowest part of the spinal canal. After birth the bony structures grow more rapidly than the neural tissue, and the inferior end of the spinal cord gradually retracts from the lower part of the spinal canal, so that, in adult life, the spinal cord extends only as far as the first or the second lumbar vertebra. Below this the spinal canal is occupied by the cauda equina, which consists of the spinal nerves. On the anterolateral and the posterolateral aspect of each half of the spinal cord numerous fibers appear that coalesce to form small cords. There are thirty-one of these on each side, and they are called the spinal nerves.

Upon cutting into the brain or spinal cord we perceive that this tissue is not homogeneous, but that the appearance differs in various places. Upon microscopic examination, we find this difference to be due either to the absence or to the preponderance of the cells, fibers, blood-vessels, and pigment. Investigations as to the relative importance, function, and relationship of these various elements have extended over many years, and as a result of these a theory, known as the "neuron theory," has been propounded.

THE NEURON

"The neuron is a nerve-cell, and all of its processes, including its protoplasmic process or dendrons, and the single axis-cylinder

process with its cone of origin, its collaterals or side branches, and its terminal arborizations.

"The neuronic doctrine is that the nervous system consists of innumerable anatomically independent nervous units in contiguity, but not in continuity. There are interlacings of processes, but no network; the nervous units are genetically and trophically independent cellular organisms, arranged in functionally correlated systems and communities" (Mott).

With this conception of the nervous system, it becomes necessary to study, first, the neuron, its construction and function; second, groupings of these neurons into systems, communities, and constellations; and, finally, the associative interactivity of the neurons and collections of the neurons.

A neuron is a nerve-cell and all its processes considered as a structural unit. The cell-body varies in size from $\frac{1}{3500}$ to $\frac{1}{250}$ inch. Neurons vary in shape, being irregularly spheroid, pyramidal, spindle shaped, or globular. They give off one or more processes, and may be classified as unipolar, bipolar, or multipolar neurons. In most cells one of the processes is continued a good distance, and is generally known as the axon and axis-cylinder process. The other processes are usually shorter, branch, and may be very tortuous; they are known as dendrons.

The cell-body is composed of a semifluid albuminous substance, the cytoplasm. Within this lies a nucleus, and within this again a nucleolus. The cell-body is not homogeneous, but is traversed by a fine network of fibrillæ that pass from the dendrons through the cell-body, where they interlace, and out of the cell-body with the axon. Within this network there are fine granular bodies known as Nissl's bodies, or chromophile substance. These bodies are arranged in quite a definite manner around the nucleus.

The *dendrons* are branching protoplasmic substances that resemble the cell-body in structure. As they extend they divide and subdivide, resembling in appearance the root of a tree. In some cells they are very numerous and terminate in fine fibrils. They may also present on their surface small nodules called gemmules, or buds. Within the dendrons are fine fibrils that join the fibrillar network of the cell-body (Fig. 5).

The *axon* or *axis-cylinder process* is always single, arising from

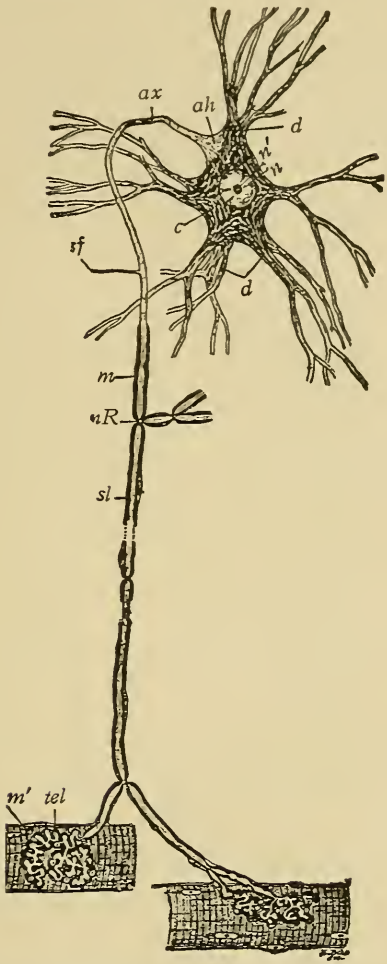


Fig. 5.—Diagram of a neuron motor from the ventral column of gray matter in the spinal cord. The cell-body, dendrites, axon, collateral branches, and terminal arborizations in muscle are all seen to be parts of a single cell and together constitute the *neuron: ah*, Axon hillock free from chromophilic bodies; *ax*, axon; *c*, cytoplasm of cell-body containing chromophilic bodies, neurofibrils, and other constituents of protoplasm; *d*, dendrites; *m*, myelin (medullary) sheath; *m'*, striated muscle-fiber; *n*, nucleus; *n'*, nucleolus; *nR*, node of Ranvier where the axon divides; *sf*, collateral branch; *sl*, neurilemma (not a part of the neuron); *tel*, motor end-plate. (After Barker.)

a cone-like projection of the cell-body that contains little or no chromophile substance, but that does contain many fine fibrils.

In the central nervous system the axon gives off numerous fine collateral branches at right angles to its course, but after it emerges from the central nervous system it does not branch until it reaches its termination, where it ends in a plexus, muscle-plates, tuft, or tassel. The axon is made up of many fine fibrils that are continuous with those of the cell-body and the dendrons. When the axons leave the gray matter they are insulated from one another by receiving a fatty coating or medullary sheath, which is held in place by a thin membrane; this they lose again at their termination.

A second type of cells develops outside of the central nervous system, in the ganglia, on the posterior roots; they are bipolar, but during development the two processes approach each other and then appear to be fused for a short distance. Thus in the adult, this type of neuron is pear shaped, and appears to have but a single process, which diverges into two parts, the one forming the sensory portion of the peripheral nerve and terminating in brush-like expansions on the skin, in plexuses, and bulbs of Krause, or tactile corpuscles; the other enters the spinal cord through the posterior roots and also terminates in brush-like expansions.

From what has been said we see that a neuron is made up of a cell-body, with the nucleus, nucleolus, reticulated fibrillæ, chromophile substance, and a number of processes, one of which is called an axon, the others being termed dendrons.

Both axons and dendrons are made up of a number of fibrillæ surrounded by myelin, this in turn being invested in a sheath. Although the fibrils are continuous, we find that the myelin sheath consists of a series of short tubes, from 1 to 1.5 mm. long. The point of junction of the two tubes is indicated by a constriction known as the node of Ranvier. The external sheath, called the sheath of Schwann, is a connective-tissue structure, and in the adult fiber is continuous over the node of Ranvier. The sheath of Schwann is not present in the central nervous system. Nerve-fibers lose the myelin substance and also the sheath of Schwann at their peripheral termination.

A nerve-fiber is made up of central conducting strands, which are insulated by a series of cylinders placed end to end, myelin

substance, and covered by the sheath of Schwann. Such fibers are called medullated nerve-fibers, and constitute the principal part of the peripheral nervous system. The non-medullated fibers, or fibers of Remak, are made up only of fibrils surrounded by a neurilemma sheath. They present a grayish appearance. The sympathetic and the olfactory nerves consist chiefly of non-medullated nerve-fibers (Fig. 6).

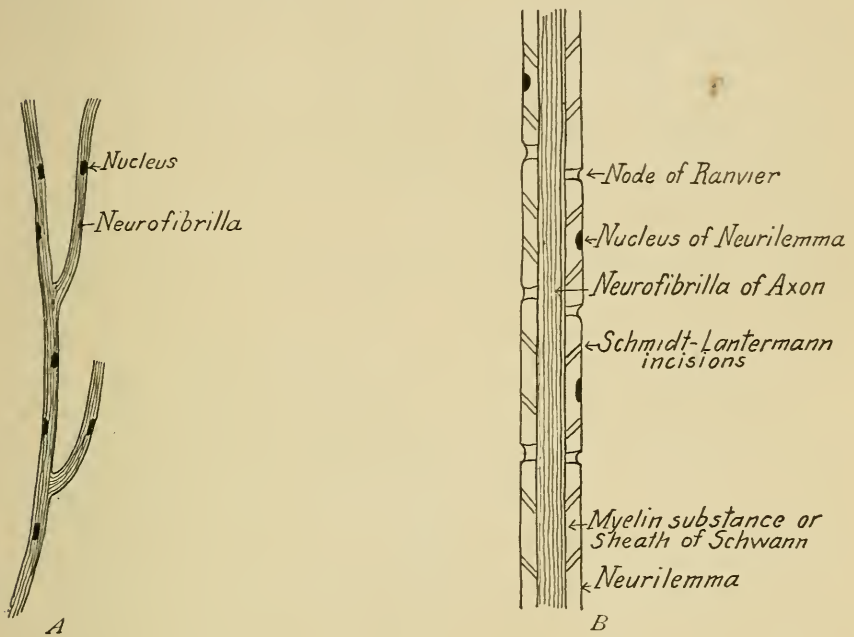


Fig. 6.—Longitudinal section: *A*, Non-medullated fiber from the sympathetic system; *B*, medullated fiber. (Diagrammatic.)

Function of the Cell.—Waller, Türcke, von Gudden, and many other observers have shown that whenever a process is separated from its cell-body the process degenerates. On this fact was formulated the Wallerian law of degeneration, which is the basis of much experimental and pathologic investigation regarding the course of nerve-tracts and the origin of nerve-fibers. This law holds that whenever a cell process is separated from its cell-body, the distal portion of the separated process degenerates throughout its extent and becomes functionless. Changes also take place in the proximal portion for a short distance, and minor

changes occur in the cell-body. This degenerative process begins almost immediately or within a few hours, and may be demonstrated microscopically.

The Wallerian law proves that one of the functions of the cell is to provide nourishment for the processes connected therewith. This is further proved by the fact that if a cell is destroyed, either experimentally or as the result of disease, all its processes degenerate throughout their extent and are rendered functionless. It has also been shown experimentally, and as the result of clinical investigation, that so long as the cell is intact and other certain conditions are present, the proximal portion of the divided process begins to grow and send out fibrillæ, which, under proper conditions, will cause regeneration of the distal portion of the divided process. Nerve-fibers, as we have seen, are capable of conducting impulses in both directions, but physiologically they conduct them only in one way. There must, therefore, be some activity of the cell-body that permits impulses to travel in only one direction. Moreover, the cell-body of the neuron must have a still higher and more important function, that is, the power of selective and discriminating activity. It must be an inherent function of the nerve-cell to be able not only to determine the direction of the impulse, but also to direct impulses to their proper destination.

It will be seen, therefore, that the cell-body possesses the following functions: It nourishes its process, that is, it is the trophic center, and, under proper conditions, it causes its process to regenerate; it governs the direction of impulses; it determines the destination of and directs all outgoing impulses. How this determination of the destination and the direction of the impulse to its proper station are accomplished is a question that is inextricably bound up with the problem of life itself.

The mode of transmission of stimuli from one cell to another in the human being is still a disputed question. Whether the processes are capable of ameboid movement and can thus produce contact when desired, as is believed by some; whether a physiologic change occurs in the material through synapses that separate the process of one cell from the processes or cell-body of another, thus allowing the transmission of the impulse

from neurofibrils of one cell to those of another—these are still moot points.

If one understands the function of a single neuron, further comprehension of the activities of the nervous system resolves itself into a knowledge of the grouping together of numbers of neurons, the effect of one transmitting its activity to another or to others, and the result of this activity, as expressed in one of the physiologic functions of the body. Thus disease of the nervous system is an abnormal functioning of one or more neurons, expressed in abnormal reaction.

ANATOMY AND PHYSIOLOGY OF A PERIPHERAL NERVE

There is no histologic difference between a sensory, a motor, and a mixed nerve. The only difference between these varieties

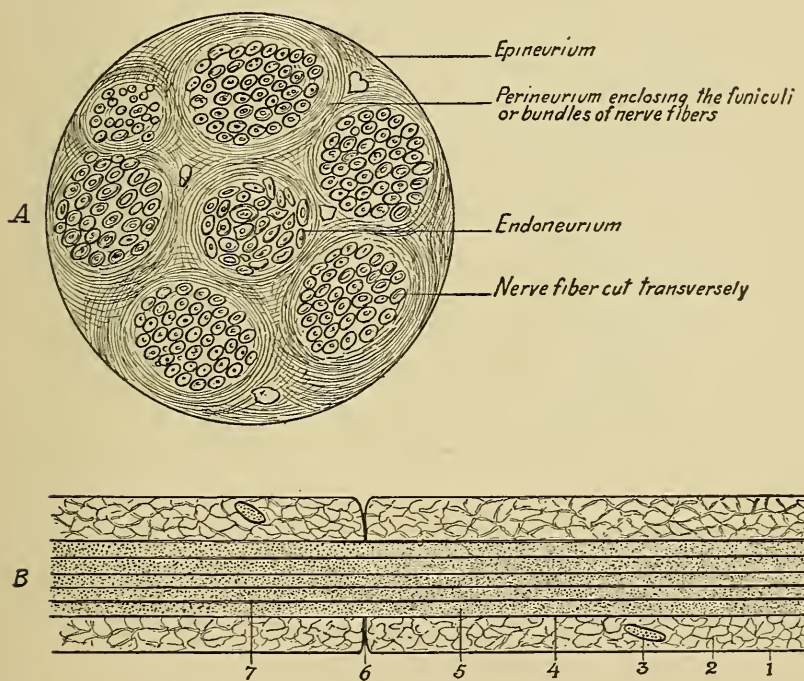


Fig. 7.—*A*, Transverse section of a nerve. *B*, Longitudinal section of a nerve: 1, Primitive sheath or neurilemma; 2, medullary sheath or white substance of Schwann; 3, nucleus; 4, axilemma (of Kuhne); 5, neuroplasm; 6, node (of Ranvier); 7, fibrillæ (of Schultze).

is that the motor nerves have a special form of ending, which will be described later, whereas the sensory nerves have a special form of origin, which will also be discussed further on.

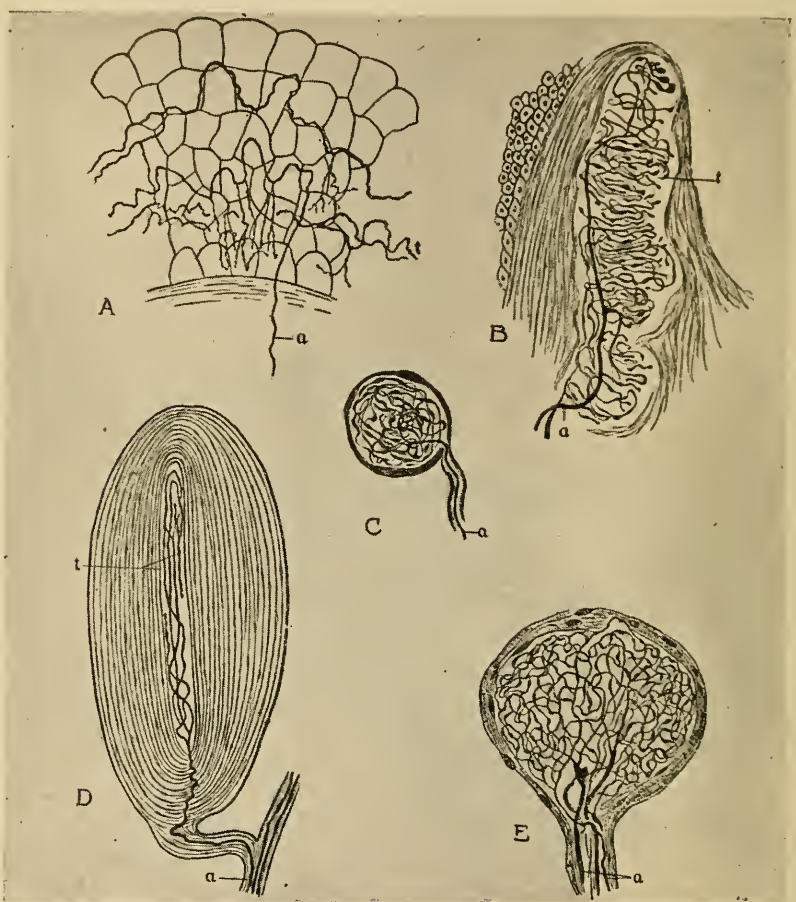


Fig. 8.—Showing some varieties of peripheral terminations of afferent neurons (or "peripheral nerve beginnings."): *A*, Terminal fibrillæ in epithelium (after Retzius); *B*, tactile corpuscle (Meissner's, after Dogiel); *C*, bulbous corpuscle (Krause's, after Dogiel); *D*, lamellated corpuscle (Pacini's, after Dogiel, Sala, and others); *E*, genital nerve corpuscle from human glans penis (after Dogiel): *a*, axon; *t*, telodendria.

A peripheral nerve is made up of a great number of conducting strands that are separated from one another by specialized substances. On its surface a peripheral nerve presents a white and

glistening sheath, called the *epineurium*. Upon cross-section of a nerve and examination under a microscope it will be seen that the nerve is divided into numerous large bundles or fibers that are separated from one another by the *perineurium*. This perineurium supports the small blood-vessels of the nerves. The large bundles are divided in turn into smaller bundles, which are separated from one another and surrounded by endoneurium.

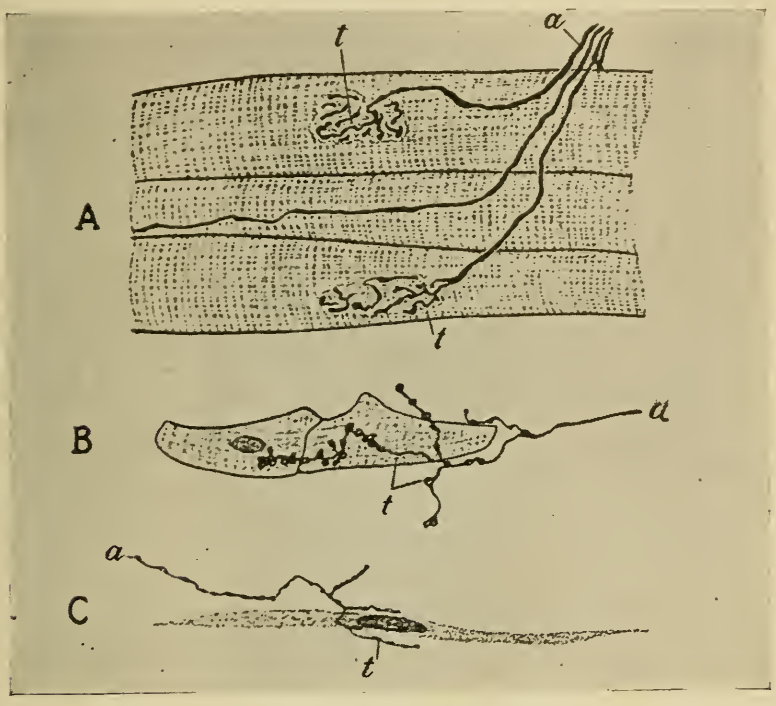


Fig. 9.—Diagram of motor nerve-endings: *A*, Striated muscle; *B*, cardiac muscle; *C*, unstriated muscle: *a*, axon; *t*, telodendria. (After Huber, Böhm and Davidoff, and others.)

These smaller bundles are seen to consist of numerous axis-cylinder processes or axons, each of which is surrounded by the sheath of Schwann and the myelin substance. When one of these axis-cylinder processes is examined under high magnification, it is seen to consist of numerous fibrils that are held together by a cement-like substance called the neurilemma (Fig. 7).

Motor nerves terminate in muscle plates, in plexuses, or in specialized endings; sensory nerves originate at the tactile cor-

puscles in the bulbs of Krause, plexuses, tufts, or in specialized endings (Figs. 8, 9).

Nerve-fibers are capable of conducting stimuli in both directions, and transmit these at the rate of about 90 feet a second. Although, as stated, nerve-fibers are capable of conducting stimuli in both directions, yet physiologically a normal stimulus is transmitted only in one direction: in the axis-cylinder processes, away from the cell; in the dendritic processes, toward the cell.

ANATOMY AND PHYSIOLOGY OF THE SPINAL CORD

The spinal cord is that portion of the nervous system contained in the spinal canal of the vertebral column. It extends from the foramen magnum to the coccyx. It is cylindric in shape and flattened anteroposteriorly. It has two enlargements—one above, known as the cervical enlargement, and one below, known as the lumbar enlargement. The average diameter of the spinal cord in the adult is about $\frac{1}{2}$ inch anteroposteriorly, and slightly greater laterally. It is covered by the dura mater, which is continuous with that which invests the brain, is not adherent to the bony walls of the vertebræ, but is separated from them by loose areolar tissue and a plexus of veins. The cord extends as low as the third sacral vertebra; beyond this it is continued as a slender cord to the back of the coccyx, where it blends with the periosteum. The sheath is much larger than is necessary for its contents. Its inner surface is smooth, and on each side openings may be seen that transmit the two roots of the corresponding spinal segment, the dura being continued in the form of a tubular prolongation on them as they pass through these openings (Figs. 10, 11).

On opening the lower part of the dura mater the roots of the lumbar and sacral nerves are brought into view. These roots, taken together, form the cauda equina. The dura is made up of fibrous and elastic tissue, the internal surface being covered by endothelial cells. It is but sparingly supplied with vessels and has few nerves.

The *arachnoid* is a thin, delicate membrane that invests the

surface of the cord. It is connected with the pia by slender connective-tissue filaments. It is continuous with the cerebral arachnoid, and is reflected in the form of a sheath over the various nerves as they pass outward to the intervertebral foramen. The outer surface of the arachnoid is in contact with the inner surface of the dura, and in places is connected with it by means

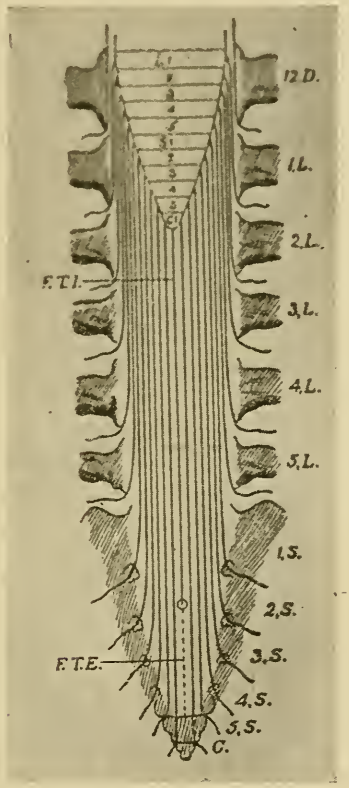


Fig. 10.—Cauda equina (Fleming, in Allbutt's "System of Medicine").

of connective-tissue trabeculæ. Especially does this occur at its posterior portion, where the fasciæ dentatæ are formed.

The inner surface is separated from the pia by a considerable space, called the *subarachnoid space*. This space is largest in the lower part of the spinal canal, where it incloses the cauda equina. Superiorly it is continuous with the cranial subarachnoid space, and communicates with the general ventricular cavity of the

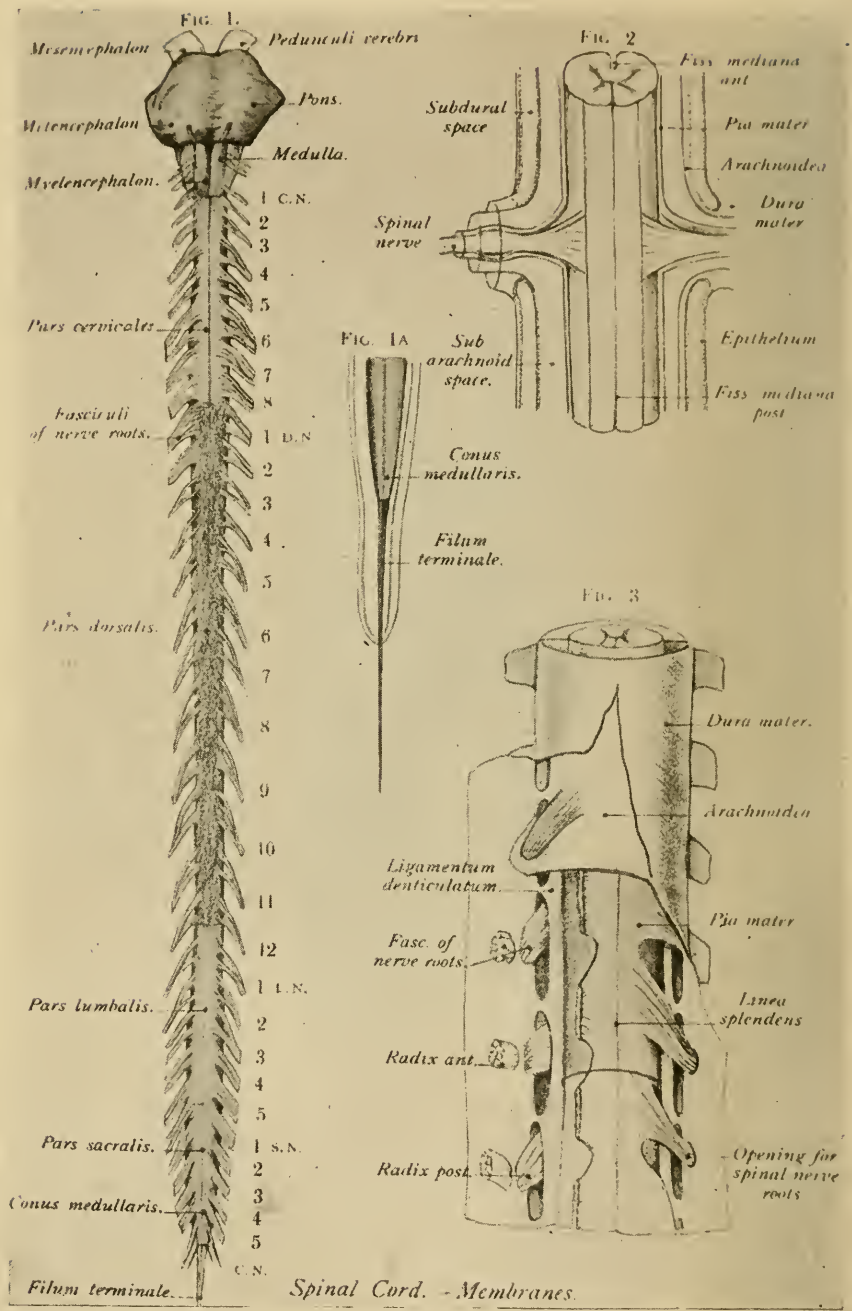


Fig. 11.—Membranes of the spinal cord (Whitaker, "Anatomy of the Brain and Spinal Cord")..

brain by means of openings in the pia mater at the inferior boundary of the fourth ventricle by the foramina of Magendie and of Retzius.

The subarachnoid space is filled with an abundant serous secretion, the cerebrospinal fluid, and allows a certain amount of motion to the spinal cord.

The *pia mater* covers the entire surface of the cord, to which it is intimately adherent, and sends processes downward into the anterior fissure. It forms a sheath for each of the filaments of the spinal nerves, and invests the nerves themselves. It contains a number of lymphatic spaces that communicate with the subarachnoid space. The pia is well supplied with blood-vessels, which are inclosed in perivascular sheaths derived from its inner layer. It receives its nerve supply from the sympathetic plexus.

The spinal cord is about 17 or 18 inches long, and weighs about one and one-half ounces. It does not nearly fill the vertebral canal. In the adult it occupies the upper two-thirds of the canal, extending from the upper border of the axis, where it is continuous with the medulla, to the lower border of the body of the first lumbar vertebra, where it is terminated in a slender filament of gray substance, which is continued for some distance as the filum terminale. In the three-months' fetus it extends to the bottom of the sacral canal, but as the fetus grows older it gradually recedes from below. This is due to the fact that the growth of the bones comprising the canal is proportionately more rapid than that of the cord. At birth the cord extends as far as the third lumbar vertebra (Fig. 12).

The cervical enlargement extends from the third cervical to the first or second dorsal vertebra. Its greatest diameter is laterally. The lumbar enlargement is situated opposite the last two or three dorsal vertebræ, and has its greatest diameter anteroposteriorly.

The cord presents on its anterior surface, in the midline, a longitudinal fissure,—the *anterior median fissure*,—and on its posterior aspect another fissure, not so well marked as this—the *posterior median fissure* or sulcus. The anterior median fissure extends into the cord for about one-third of its thickness, and contains a prolongation of the pia mater. Its floor is formed by

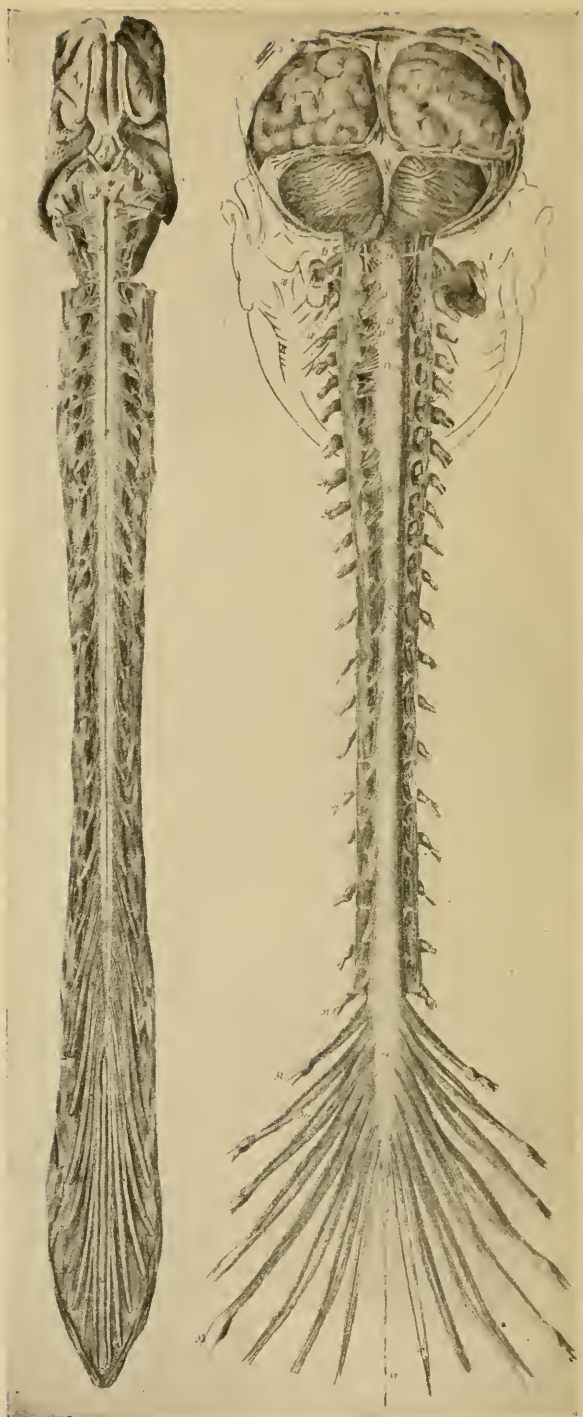


Fig. 12.—Spinal cord in the vertebral canal (Feigel).

the anterior white commissure, and it is perforated by numerous blood-vessels passing into the center of the cord. The posterior median fissure is not a depression or opening, but is a connective-tissue septum that divides the posterior part of the cord into halves and supports the blood-vessels. It extends into the cord for one-half its depth (Fig. 13).

The surface of the cord is further divided into columns by the line of emergence of the anterior roots and the depression of

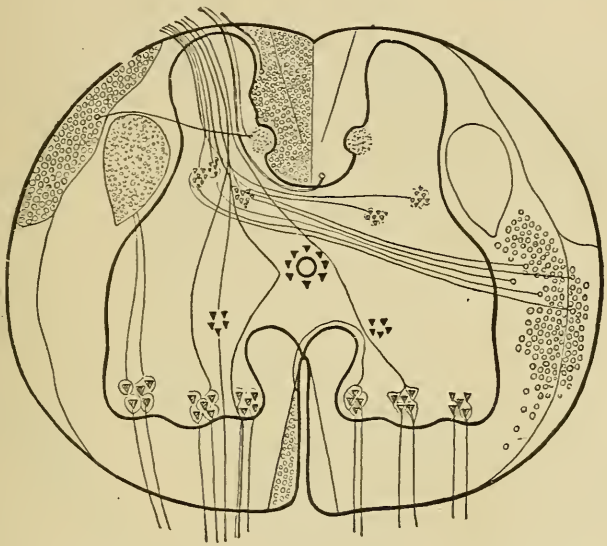


Fig. 13.—Cross-section of spinal cord, showing cells, fibers, and tracts (diagrammatic).

entrance of the posterior roots—anterior columns, lateral columns, and posterior columns (Fig. 14).

On either side of the cord, at the junction of the anterior third with the posterior two-thirds, and at the junction of the posterior third with the anterior two-thirds, there emerge small fibers. Those that emerge at the anterolateral aspect coalesce at the various levels of the cord to form a single nerve, each one being called an anterior nerve-root. This occurs also in the posterolateral aspect, each being here called a posterior nerve-root. There are 31 anterior and 31 posterior nerve-roots on each side of the cord.

A segment of the spinal cord is made up of white and of gray matter, of blood-vessels, and of a pair of spinal nerves that pass to a definite part of the body. Each segment is joined to other segments by means of nerve-tracts. Each spinal nerve is made up of two roots—the sensory and the motor. On the posterior root, within the intervertebral foramen, just before it is joined by the anterior root, there is an enlargement called a posterior root ganglion.

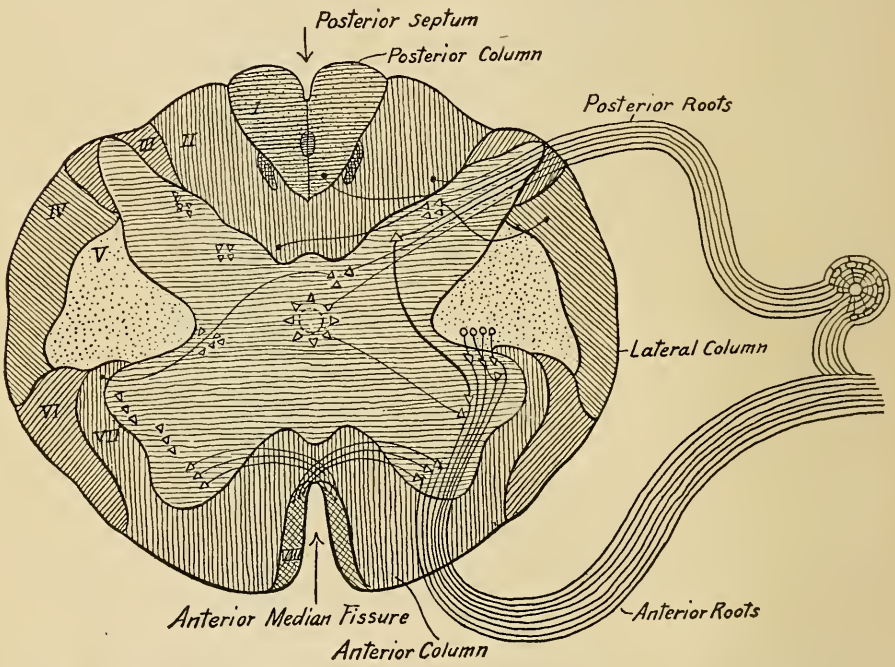


Fig. 14.—Showing columns of cord, anterior and posterior roots, etc. (diagrammatic).

The anterior and posterior roots on each side from a spinal segment coalesce in the intervertebral foramina. From this point outward they run their course as a single mixed nerve.

In the cervical, lumbar, and sacral regions the spinal nerves intermingle to form the cervical, brachial, lumbar, and sacral plexuses. There are eight (8) cervical segments, twelve (12) dorsal segments, five (5) lumbar segments, five (5) sacral segments, and one (1) coccygeal segment.

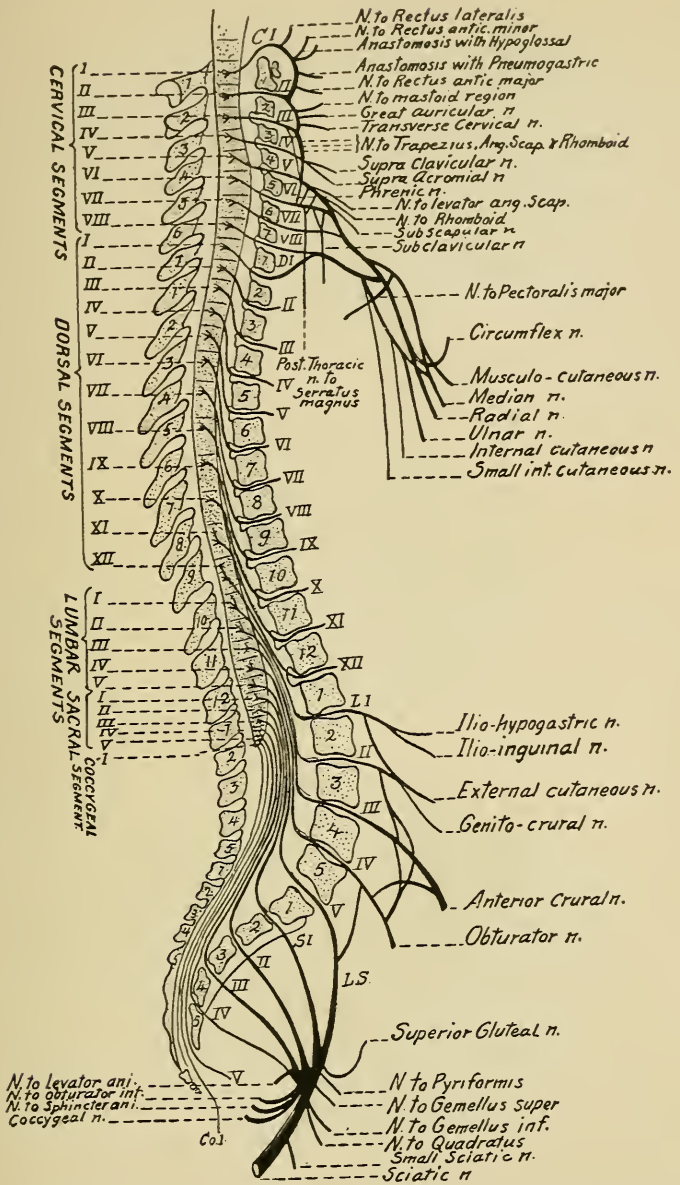


Fig. 15.—Relation of the spinal segments and nerves to the bodies and spines of the vertebra. (After Butler.)

Owing to the fact that the spinal cord itself is not so long as the spinal canal, the various segments of the cord in the lower

cervical, dorsal, and lumbar region are not directly opposite the corresponding vertebræ of the spinal column.

Chippault has given us a practical set of rules for determining the relations of the segments to the spinous processes of the vertebræ. He says: "In the cervical region add one (1) to the number of vertebræ, and this will give the segments opposite. In the upper dorsal region add two (2); from the sixth to the eleventh dorsal vertebræ add three (3). The lower part of the eleventh dorsal spinous process is opposite the three (3) lower lumbar segments. The twelfth dorsal spinous process and the space below it are opposite the sacral segments." The spinal cord ends opposite the first lumbar vertebra or opposite the cartilage in the space above it (Fig. 15).

STRUCTURE OF THE SPINAL CORD

If a transverse section is made through the spinal cord, it will be found to consist of white and of gray matter. The white matter is situated externally, surrounding the gray matter. The relative proportion of gray and of white matter varies in different regions of the cord (Fig. 16).

Upon cross-section the gray substance presents two crescentic masses, one situated in each lateral half of the cord, united by a transverse band of gray matter. Each crescentic mass presents, for purposes of description, an anterior and a posterior portion, called the *anterior* and the *posterior horn*, and in the cervical region a lateral extension on the level with the gray connecting band, called the *lateral horn*. The gray connecting band, which is called the gray commissure, contains a small central canal.

The posterior horn approaches close to, but does not touch, the surface of the cord at the posterolateral fissure, where the posterior roots enter the cord. The anterior horn does not approach the surface of the cord, but is separated from it by a definite layer of white matter.

Description of the Gray Substance.—The gray substance is made up of nerve-fibers of variable length, but of smaller average diameter than those of the white substance, of nerve-cells of various sizes and shapes, of neuroglia, and of blood-vessels.

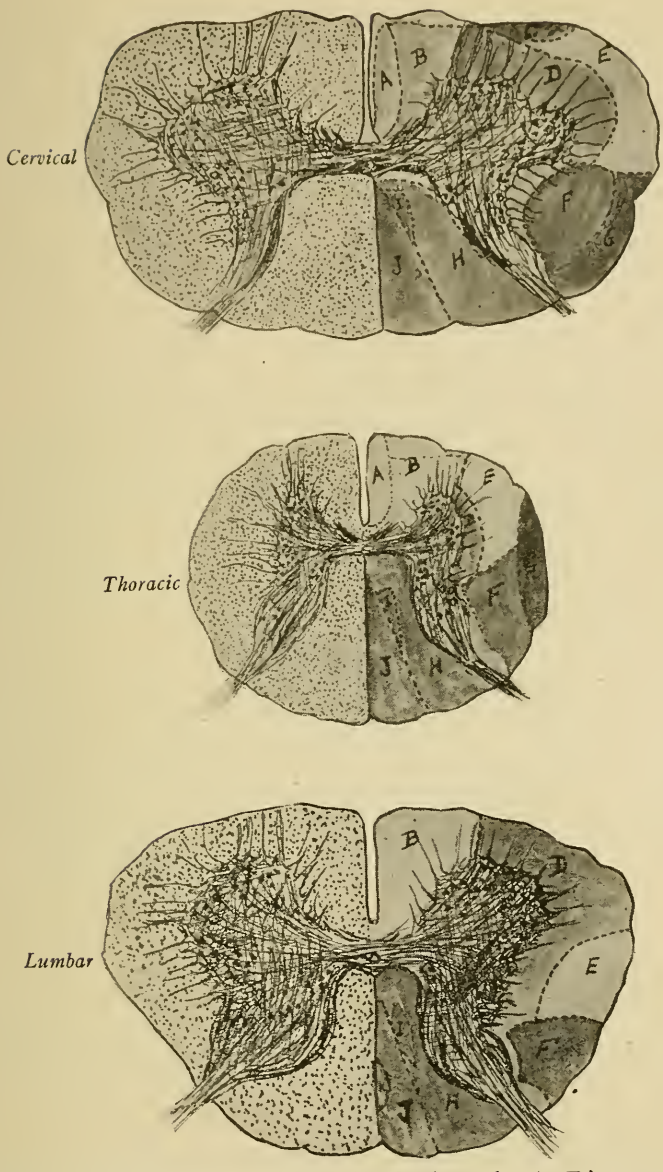


Fig. 16.—Transverse sections of spinal cord: *A*, Direct pyramidal; *B*, ventral ground-bundle; *C*, Spitzka-Lissauer's; *D*, ventrolateral ground bundle; *E*, Gowers'; *F*, crossed pyramidal; *G*, spinocerebellar; *H*, Burdach's; *I*, Schutze's comma; *J*, Goll's. (Gray's "Anatomy.")

The nerve-cells are of two kinds, large and small. The anterior horns contain groups of large cells, occupying a more or less

constant position, and arranged in the medial, anterior, and lateral portions. The cells situated in the gray commissure, around the central canal, and in the posterior horns, are, as a rule, small and irregular in shape. On the inner aspect of the posterior horn, near the gray commissure, extending from the seventh cervical to the third lumbar segment, are groups of cells arranged in a tract called Clarke's column or vesicular column.

The *posterior horn* consists of two parts, the caput cornu, surrounding the gelatinous substance, and the cervix cornu, which is the constricted portion, and connects the posterior horns with the remainder of the gray substance.

The *anterior horns* are best developed in the cervical and lumbar regions, where they give origin to the nerves of the extremities, and contain more or less well-marked and constantly situated groups of large cells.

Extending from the gray matter opposite the gray commissure in the cervical and upper dorsal region is a lateral extension of gray matter called the *lateral horn* or cornu.

The *gray* or *posterior commissure* is that portion of the gray substance which connects the two crescentic masses. It is separated from the bottom of the anterior median fissure by the anterior white commissure. Extending through the gray commissure and running the entire length of the cord is a minute canal which is barely perceptible, in the human being, to the naked eye. This canal is called the central canal, and opens into the fourth ventricle. Grouped irregularly around this canal throughout its entire length are numerous small cells.

Neurogliar tissue is made up of small, stellate or branched cells called glia-cells, and of a network of very delicate fibrillæ. This tissue forms the ground-substance in which nerve-cells, their processes, and blood-vessels are embedded. A considerable amount of this tissue is accumulated on the surface of the cord beneath the pia, around the central canal, and in the posterior part of the posterior horn.

The White Substance.—This is made up of longitudinal fibers arranged in columns, of oblique and transverse fibers.

The White Commissures.—The anterior white commissure, situated in front of the gray commissure, and separating it from

the anterior median fissure, is made up of fibers that cross from the anterior columns of one side to end in the anterior horns of the opposite side. The posterior white commissure is a small band of transverse fibers situated immediately posterior to the gray commissure.

The oblique fibers are present at all levels of the cord. They are the fibers that enter or leave the longitudinal columns, to terminate or originate in the gray matter. The longitudinal

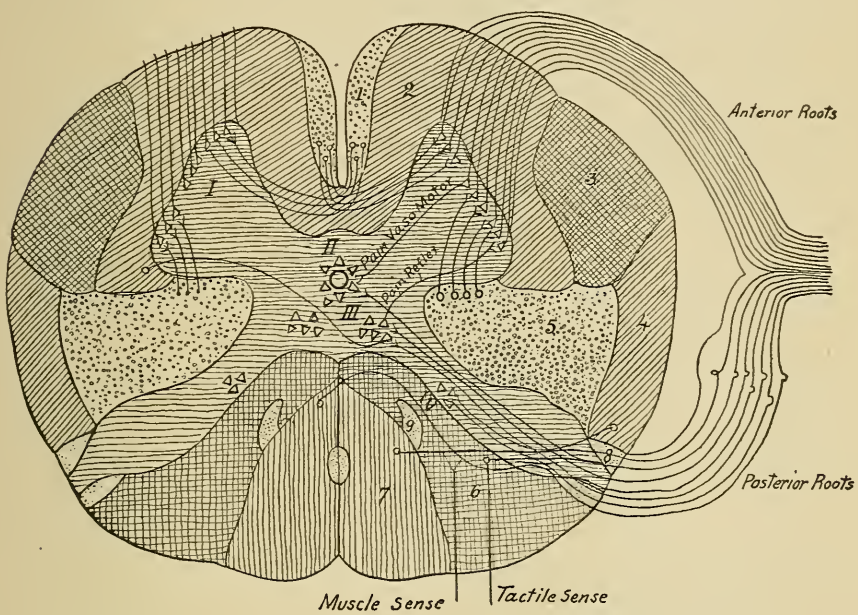


Fig. 17.—1, Direct pyramidal tract; 2, anterolateral ground-bundle; 3, Gowers' tract; 4, direct cerebellar tract; 5, crossed pyramidal tract; 6, Burdach's; 7, Goll's; 8, Lissauer's tract; 9, comma tract; I, anterior horn cells, II, cells around central canal; III, cells at anterior part of posterior horn; IV, Clarke's column of cells. Diagrammatic.

fibers pursue a longer or shorter course, and either originate or terminate in the gray matter (Figs. 17 and 19).

Anterior Roots.—The axons of the large cells in the anterior horn emerge through the anterior horn, pierce the anterior column, issue from the spinal cord as the anterior roots, and form the motor portion of the peripheral nerve.

Processes from the cells around the central canal emerge with

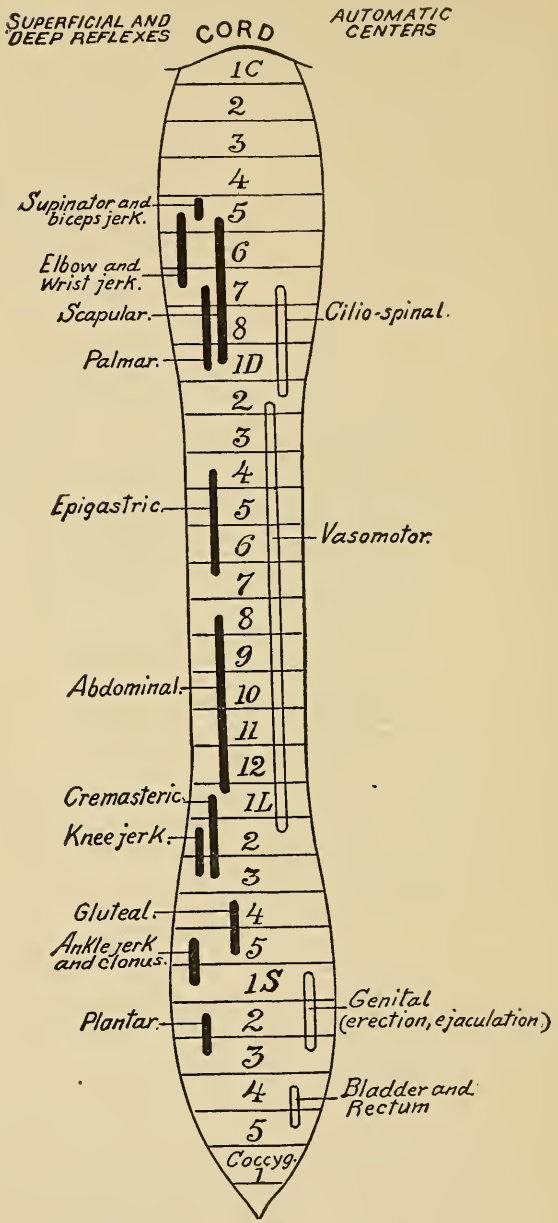


Fig. 18.—Location of centers of spinal cord.

the anterior and the posterior roots. Processes of cells in the anterior part of the posterior horn cross the cord in the gray

commissure, and ascend in the anterolateral ground-bundle, or, without crossing, ascend in the anterolateral ground-bundle of the same side. Processes from cells in Clarke's column enter the direct cerebellar tract of the same side and ascend the cord.

Processes from cells in the posterior horn traverse the gray matter and end around cells in other parts of the gray matter, either on the same or on the opposite side, above or below.

Posterior Roots.—Fibers from the posterior roots enter the cord and ascend for a short distance along Lissauer's tract. They then enter the posterior horns, where they may end around cells in the anterior portion on each side, around cells near the central canal, around cells in the posterior portion, around cells in Clarke's column, around cells in the anterior horn of the same or the opposite side etc. Some fibers enter the column of Goll and Burdach's column on the same or on the opposite side, divide, and ascend and descend (Figs. 17 and 19).

In some regions of the spinal cord collections of cells occur that seem to have definite functions. These cells are called centers. The most important of these centers are found in the sacral region of the cord. Here we have the rectal, vesical, and genital centers. Grouped around the central canal throughout the course of the cord are the vasomotor centers. At about the junction of the first dorsal and lower cervical are the ciliospinal centers (Fig. 18).

Important Columns and Fiber-tracts of the Spinal Cord (Figs. 13, 14, and 17).—The spinal cord is separated by means of anterior fissures, anterior roots, posterior roots, and the posteromedian sulcus into anterior, lateral, and posterior columns. Between the anterior roots and the anterior median fissure is the anterior column—one to each half of the cord. Between the anterior roots and the posterior roots is the lateral column—one to each side of the cord. Between the posterior roots and the posteromedian sulcus is the posterior column—one to each half of the cord.

Each of these columns is further subdivided into tracts. A knowledge of the origin, course, and termination of these tracts has been ascertained as the result of embryologic development, physiologic experimentation, and clinicopathologic observation.

The anterior column contains an important tract, called the *direct pyramidal tract*, or the fasciculus of Türck, which is continuous with the uncrossed fibers of the pyramids of the medulla. This tract is most marked in the cervical region, gradually becoming smaller and disappearing in the lumbar region.

In the lateral column, just in front of the posterior horns, and about midway between the lateral surface of the cord and the gray commissure, is a more or less oval-shaped tract, known as the *crossed pyramidal tract.* This is continuous with the larger portion of the pyramid, descending from the opposite side of the medulla.

Occupying the posterior half of the lateral aspect of the cord upon its outer circumference is a collection of fibers termed the *direct cerebellar tract.* This tract, when traced upward, is found to pass into the inferior cerebellar peduncle of the same side.

Anterior to this pathway, and occupying the anterior half of the lateral circumference of the cord, as far forward as the anterior roots, is the *anterolateral ascending cerebellar* or *Gowers' tract.* Between the posterior horns and the circumference of the cord is a small area through which fibers ascend or descend for a short distance before entering the posterior horns; this is called *Lissauer's marginal tract.*

In the posterior column, immediately adjacent to the posterior horn, is a collection of fibers known as Burdach's column, or the fasciculus cuneatus. Adjacent to the column of Burdach, and extending to the posterior median septum, is another group of fibers, known as the column of Goll, or the fasciculus gracilis. Between the column of Goll and the column of Burdach is a small tract termed the *comma tract* or the comma-shaped bundle.

In the lateral columns, extending also into the anterior columns, are numerous fibers that originate in cells situated in the anterior part of the posterior horns of the same or the opposite side. These fibers are interspersed with numerous other fibers situated in this region. They do not form a definitely outlined tract, but are grouped and described under the name of the *anterolateral ground-bundle.*

In addition to these tracts, which pursue a long course through the spinal cord and connect the cord with structures in the

cranial cavity, there are numerous short tracts connecting various segments of the cord with one another. There are also many fibers in the spinal cord that pursue an oblique course and connect different levels of the two sides of the cord with each other. These are called *association* and *commissural fibers.*

Origin and Termination of the Important Tracts of the Cord (Fig. 19).—The *direct pyramidal tract*, or fasciculus of Türck, is made up of 10 per cent. of uncrossed pyramidal fibers from the medulla. These fibers have their point of origin in the precentral convolutions of the frontal lobe of the brain. The direct pyramidal tracts occupy the median portion of the anterior column. At various levels in the cord they give off fibers that cross the anterior white commissure to the opposite side of the cord, where they enter the anterior horn and terminate around the large cells, or the processes of these cells, in the anterior horns.

The *crossed pyramidal tract* is made up of 90 per cent. of fibers of the pyramids of the medulla which have crossed in the lower part of the medulla. These fibers originate on the precentral convolutions of the frontal lobe of the brain. They descend in the lateral columns of the cord on the side opposite to which they originate. At various levels in the cord fibers are given off that cross through the lateral columns of the cord and the gray matter, and end around cells situated in the anterior horns.

The *direct cerebellar tract* is made up of fibers that originate in the cells in the column of Clarke of the same side. These fibers pass through the posterior horns and through the lateral columns, and enter the direct cerebellar tract, which is situated upon the external posterolateral aspect of the cord, immediately in front of the posterior roots. This tract ascends the cord, enters the inferior peduncle of the cerebellum, and probably ends in the central lobe of the cerebellum.

Gowers' tract, or the anterolateral ascending cerebellar tract, is made up of fibers whose cells of origin are situated in the posterior horns. These fibers cross through gray and white matter into this tract, which is situated in the anterolateral and external aspect of the cord, immediately behind the anterior roots. The tract ascends the cord and enters the medulla, where it assumes a more posterior and more nearly central position.

Fibers from this tract are believed to end around cells in the medulla, pons, and cerebellum, probably on the opposite side.

The *anterolateral ground-bundle*, situated partly in the lateral and partly in the anterior column, is made up of fibers that have their cells of origin situated in the anterior part of the posterior horns of both sides, the greater number by far originating on the opposite side, crossing through the gray commissure in an ascending direction. These fibers ascend in the anterolateral portion of the cord and enter the medulla, where they are more centrally placed. They terminate around cells in the corpora striata, caudate nuclei, optic thalami, and parietal lobe of the brain. In this tract are many ascending and descending fibers whose cells of origin and point of termination cannot be accurately determined.

Lissauer's marginal tract is made up of fibers whose cells of origin are in the ganglia on the posterior roots. These fibers enter the spinal cord as posterior roots, and ascend for a very short distance, entering either the gray matter to terminate around the cells or pass into the posterior columns.

The *column of Burdach*, or the fasciculus cuneatus, is made up of fibers having their cells of origin in the ganglia of the posterior roots. After entering the cord they ascend for a short distance along Lissauer's tract, and then enter the column of Burdach. This column is situated immediately adjacent to the posterior horns in the posterior column.

After entering the column of Burdach some of the fibers divide, one branch ascending, and in its course upward being displaced toward the central line, finally ending around the cells situated in the lower part of the posterior aspect of the medulla, known as the nucleus of Burdach, or the nucleus cuneatus. The other branch descends, and in its course gives off small branches at right angles that enter the gray matter of the cord to terminate around cells.

The *column of Goll* (fasciculus gracilis) is made up of fibers that have their cells of origin in the ganglion of the posterior roots. These fibers enter the spinal cord as the posterior roots, ascend a short distance along Lissauer's column, and then enter the column of Goll. This column is situated on each side of the

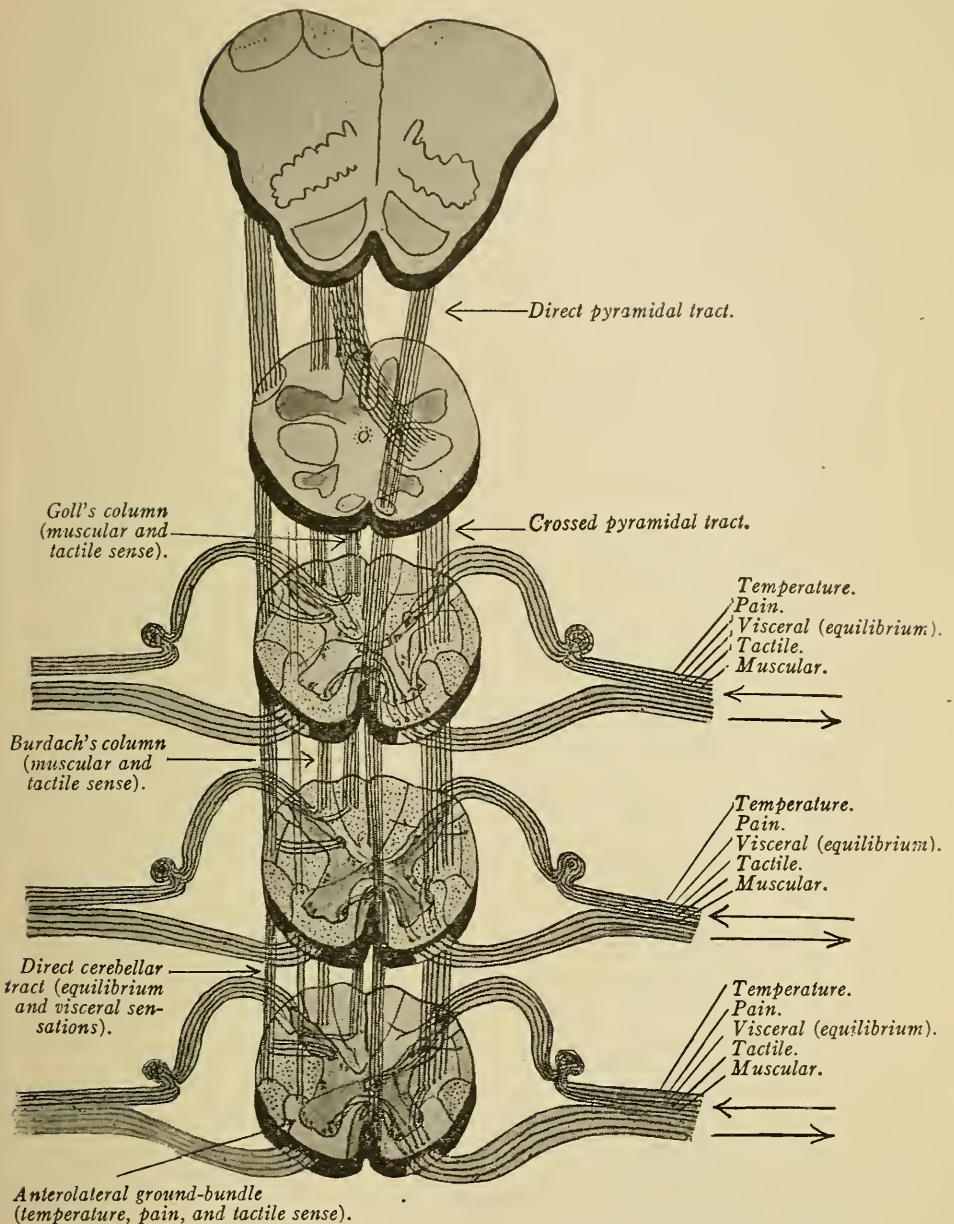


Fig. 19.—Longitudinal section of spinal cord, showing ascending and descending pathways.

posteromedian sulcus in the posterior column. Some of the fibers divide as they enter the column of Goll, one branch

ascending to end around a group of cells situated in the posterior aspect of the medulla, known as the nucleus of Goll, or the nucleus gracilis; the other branch descends and gives off in its course branches at right angles that end around cells in the gray matter. The fibers that are given off at right angles from the descending fibers of the columns of Goll and Burdach are called *collateral fibers*. Schultze's comma-shaped tract is situated between the columns of Goll and Burdach. It is made up of descending branches of fibers from the columns of Goll and Burdach.

Pathways of Ascending and Descending Degeneration.—Those tracts that have their cells of origin in the cord and transmit impulses to structures above are called *ascending pathways*, and degenerate upward. Those tracts that have their cells of origin in structures above the cord or in the cord and transmit impulses downward are called *descending pathways*, and degenerate downward. When an injury or a disease brings about a destructive lesion in the cord, all those pathways that have their cells of origin below the lesion will show degeneration of their fibers above the lesion, whereas those tracts that have their cells of origin above the lesion will show degeneration in their fibers below the affected area (Fig. 19).

BLOOD-SUPPLY OF THE SPINAL CORD

The blood-supply of the spinal cord is derived from the vertebral arteries, from the branches of the dorsal intercostals, and from the lumbars. These branches enter the spinal canal by way of the intervertebral foramina. They emerge through the dura, and some of the branches become anterior root arteries and others become posterior root arteries. These various vessels are called the lateral spinal arteries. They anastomose on the anterior surface of the cord, and form the anterior arterial plexus and the anterior spinal artery (Fig. 20). The latter extends from the medulla to the lowest part of the cord. The posterior root arteries anastomose with each other but not with those of the opposite side, and form the posterolateral arterial plexus. There are one anterior arterial plexus and two posterolateral arterial plexuses.

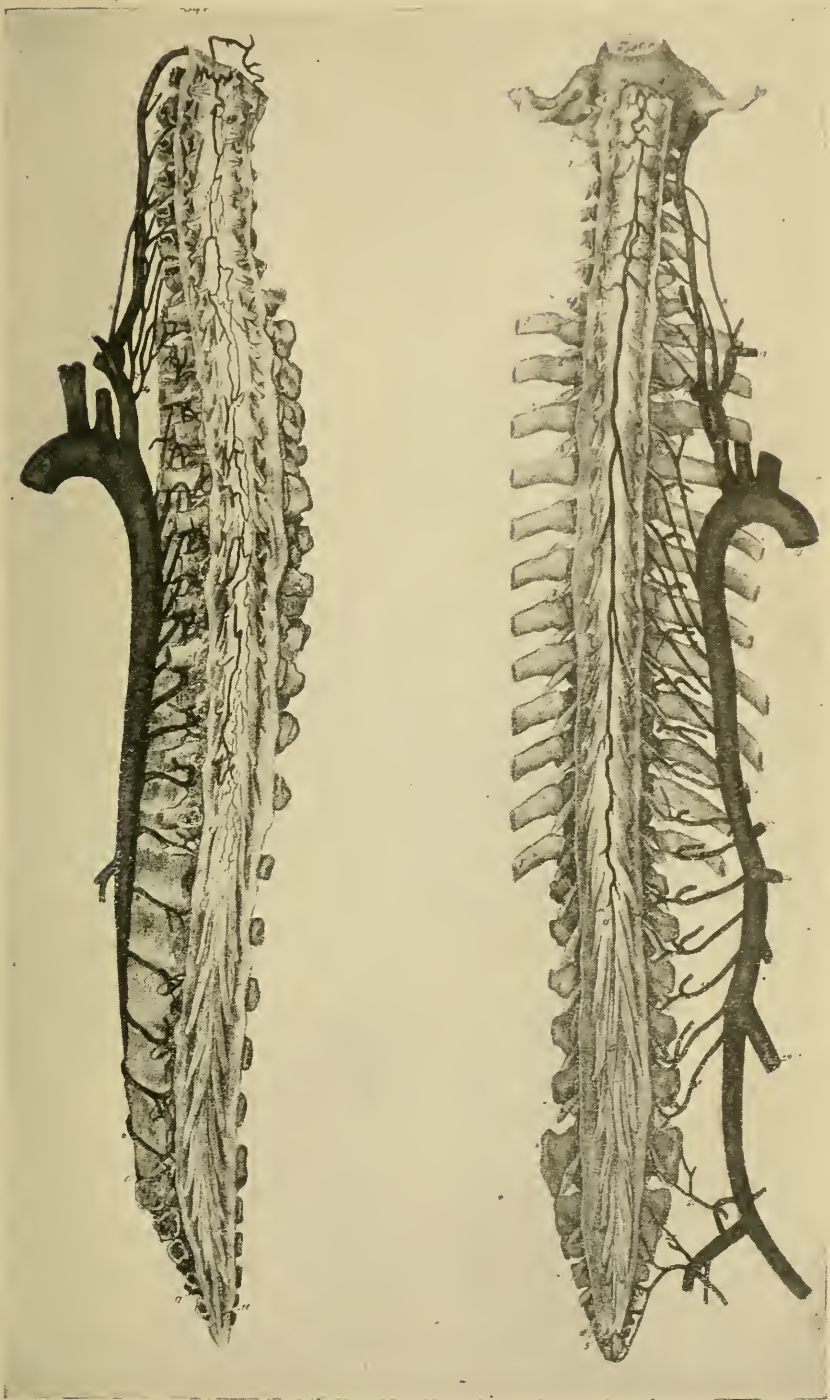


Fig. 20.—Spinal cord, showing anterior spinal artery, etc. (Feigel).

A branch arises from the anterior spinal artery which is called the anterior median artery. It is situated deep down in the anterior fissure.

From the anterior median artery branches are given off that supply the anterior horns, part of the gray commissure, the anterior

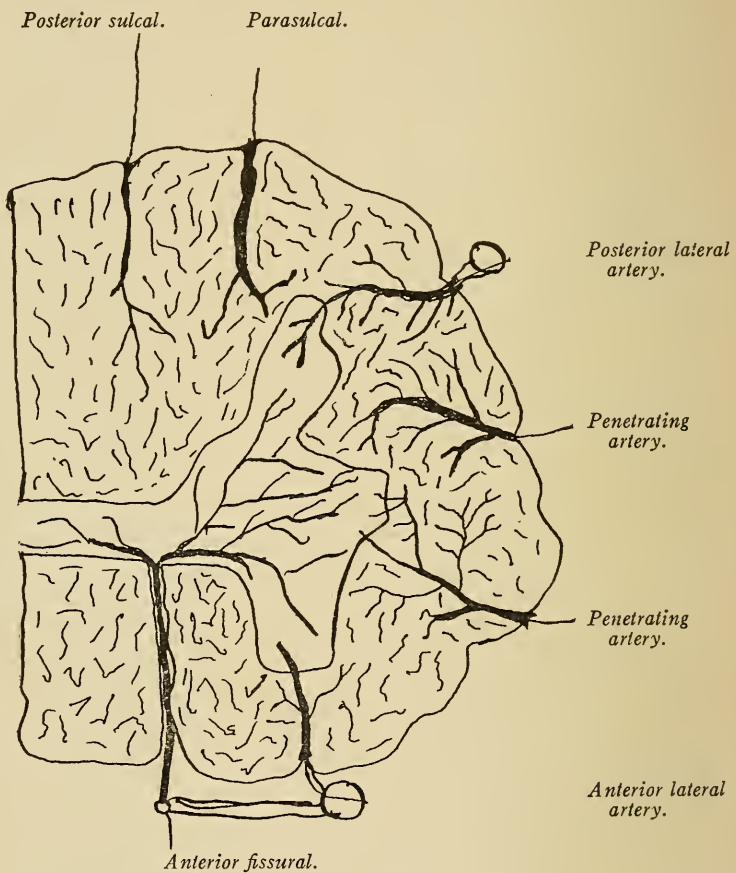


Fig. 21.—Blood-supply of the spinal cord.

portion of the posterior horns, and part of the anterior columns (Fig. 21). From the anterior arterial plexus branches are given off that supply the white matter of the anterior parts of the cord. From the posterolateral arterial plexus branches are given off that enter the cord and supply the white matter of the lateral tracts, the posterior part of the posterior horns, and the

posterior tracts. Each penetrating blood-vessel is an end-artery, *i. e.*, it does not anastomose with any other artery. The anterior spinal arteries and the anterior arterial plexus are made up of large blood-vessels that are few in number. The postero-lateral plexuses are made up of small vessels that are more numerous. The blood is collected in the capillaries, and passes up from the gray matter in the front of the cord and in front of the anterior roots into a number of minute veins. In consequence of this the blood-pressure in the gray matter of the cord, as well as in the anterior part of the cord, is high. The caliber of the arteries is large, and that of the veins is small. The blood is collected from the capillaries of the postero-arterial plexus in a number of large veins. In consequence of this the blood-pressure in the lateral tracts and in the posterior parts of the cord is low.

PHYSIOLOGY OF THE SPINAL CORD

The spinal cord exerts a more or less direct control over the various parts of the body. This control is proportional to the form of life, being most complete in the lowest forms. In addition to this function, the spinal cord is the pathway through which impulses are conducted to and from the brain.

Functions of the Important Cellular Constituents of the Spinal Cord.—The group of large cells situated in the anterior horns, which give origin to the anterior roots, preside over the muscle tone, muscle nutrition, and muscle contractility. These cells are connected with groups of muscle-fibers. When they are irritated, fibrillary muscular contractions take place; when they are stimulated, muscular contraction occurs. If individual groups of cells are stimulated, irregular and purposeless muscular contractions take place. If physiologically connected groups are stimulated, purposive muscular contractions result. These cells are constantly sending out stimuli that maintain muscle tone and normal muscle nutrition. Any interference with their normal activity results in an increase, a decrease, or a perversion of muscle contractility, muscle tone, and muscle nutrition.

The groups of small cells situated around the central canal that send their processes out through either the anterior or the

posterior roots in some way combine with the sympathetic nervous system. They terminate in the walls of the small blood-vessels. These cells preside over vascular tonicity, whereas others seem to exert their influence over the nutrition of the skin and its appendages. Still others terminate around the sweat-glands, the sebaceous glands, and in other glandular structures, and seem to influence the secretions.

Numerous small cells situated in the posterior horns serve as intermediate stations for the further transmission of sensory and coördinate impulses. Many of the small cells scattered throughout the gray matter of the cord act as commissural cells, giving origin to fibers that connect the two sides of the cord with each other. Other cells give origin to structures known as association fibers, which connect different levels of the cord with one another, and still other cells serve as intermediate stations for the transmission and wider distribution of stimuli that result in reflex activity.

In certain parts of the cord there are collections of cells that preside over definite functions of the body; these are called centers. The most important of these centers are situated in the lower lumbar and sacral region, being termed respectively the vesical, the rectal, and the genital centers. Throughout the entire course of the cord, near the central canal, are centers known as the vasomotor centers; in the upper dorsal region is the ciliospinal center, etc.

Function of the Tracts of the Cord.—The function of the tracts of the cord is to conduct upward and downward stimuli that originate in the centers above the cord, in the cord itself, or in the periphery. The tracts are divided into three groups: (1) *Projection pathways*, which transmit stimuli from the higher centers to the cord or from the periphery or from the cord to the higher centers. (2) *Association pathways*, those which connect different levels of the cord with one another. (3) *Commissural tracts*, which connect the two sides of the cord with each other.

The Functions of the Important Projection Pathways.—The important projection pathways are the direct pyramidal tract, the crossed pyramidal tract, the direct cerebellar tract, the

column of Goll and of Burdach, and the ascending anterolateral ground-bundle.

The direct and crossed pyramidal tracts transmit impulses that stimulate the anterior horn cells to activity and result in voluntary purposive motor activity. These pathways, however, more or less constantly transmit a form of stimulation that exerts an unconscious restraining or inhibiting effect upon the anterior horn cells, thereby preventing them either from receiving or from sending out impulses that result in useless reflex activity. This inhibiting effect can be increased at will. As the result of a disturbance of normal conductivity of these pathways there occurs either weakness or paralysis, with increased reflex action or abnormal motor activity, such as convulsion, contracture, muscle rigidity, etc.

Functions of the Direct Cerebellar Tract.—This pathway transmits upward that form of sensation by which equilibrium is maintained.

The Functions of the Column of Goll and of Burdach.—These tracts transmit upward muscle sense and partial tactile sense.

The Function of the Anterolateral Ground-bundle.—Many of the fibers situated in this tract transmit upward the sensation of pain, temperature sense, and partial tactile sense. Others transmit impulses upward or downward, and are secondary pathways for motion coördination, sensation, etc.

Sensation may be considered under at least three subdivisions: the epicritic sense, the protopathic sense, and deep sensibility.

The *epicritic sense* is the finer form of sensation by means of which differentiation is made between the most closely related stimuli and the most delicate forms of perception, such as slight differences of temperature, of pain, of touch, slight changes in the location of stimuli, etc.

Protopathic sensation is the coarser form of sensation. By it differentiation can be made only between the more widely separated forms of stimulation, such as marked differences in temperature, heavy touch, sharp pain, etc.

Deep sensation is that form which exists in the joints, muscles, etc., whereby changes in position of the various parts of the body to each other are recognized.

Head has shown that epicritic sensation corresponds to peripheral nerve distribution, and that protopathic sensation corresponds to posterior root distribution. The more nearly a peripheral nerve represents the supply of one posterior root, the more definite will be the outline of sensory disturbance. Conversely, the greater the number of posterior roots that enter into a nerve, the less definite will be the border of sensory disturbance, and the less marked will be the protopathic symptoms. This observer also asserts that deep sensation is transmitted by sensory filaments in the motor nerve.

Sensation may be further divided according to various qualities; *e. g.*, tactile, pain, temperature, and muscle sense.

The direct cerebellar tract, the columns of Goll and Burdach, and the anterolateral ground-bundle are the pathways through which the various forms of sensation are transmitted. These pathways do not conduct the various types of sensation indiscriminately, but each type is more or less limited to a special tract. In diseased conditions it may be possible for the various tracts to act in a compensatory manner.

The various forms of sensation are received by the peripheral nerves at their termination in the skin, muscles, joints, glandular structures, etc. They are transmitted toward the spinal cord, and the impulses then go through the ganglia on the posterior roots, and enter the spinal cord by means of the posterior roots. Upon entering the cord the pathways for the various forms of sensation diverge.

The form of sensation that is interpreted as muscle sense, or sense of position, ascends by means of fibers that enter the columns of Goll and Burdach on the same side. The stimuli that are responsible for maintenance of equilibrium, after entering the spinal cord through the posterior roots, are transmitted to cells in the column of Clarke on the same side. From these cells the stimuli are transmitted by the fibers of the direct cerebellar tract on the same side, to ascend and stimulate centers situated above. The stimuli that are interpreted as heat, cold, and pain, upon entering the spinal cord through the posterior roots, are transmitted to cells situated in the anterior part of the posterior horns. From these cells the stimuli are transmitted through

fibers that cross and ascend in the anterolateral ground-bundle of the opposite side. The stimuli that effect tactile sensation upon entering the spinal cord pursue two courses: one is similar to the muscle sense, that is, it is transmitted upward in the columns of Goll and Burdach of the same side; the other is similar to pain and temperature sense, that is, it is transmitted upward in the anterolateral ground-bundle of the opposite side.

Functions of a Segment of the Spinal Cord and Symptoms Arising as a Result of a Pathologic Condition of a Segment of the Cord.—A segment of the spinal cord is that part of the cord that gives origin to one set of spinal nerves. Each segment of the spinal cord is connected with a definite region of the body.

The function of a segment of the cord is to preside over nutrition, the tone, the vasomotor state, and the reflex activity of a portion of the body, and to allow the transmission upward and downward of stimuli to and from the higher centers.

The pathologic conditions affecting a segment may be limited in extent to the gray substance or to the white matter. The condition may be diffuse, involving both the gray and white matter on both sides, or it may be limited in extent, involving the gray and white substance in a circumscribed area limited to one side, or it may partly involve both sides. On the other hand, the lesion may be so extensive as completely to disintegrate one or more segments.

The symptoms arising from these pathologic conditions vary. Those due to interference with the gray matter manifest themselves in a loss or decrease of reflex activity, of muscle tone, of muscle nutrition, and of muscle power. There is also loss or decrease of temperature, of pain, and of partial tactile sense, and there are trophic, vasomotor, and secretory disturbances. The distribution of these symptoms corresponds with the distribution of the spinal nerves connected with the affected segment.

The symptoms due to disturbance of the tracts of the segment are weakness or paralysis, with increase of deep reflexes in the part of the body situated below the lesion, loss or disturbance of control of the rectal and vesical reflexes, and disturbance or loss of tactile, pain, temperature, and muscle sense in the parts of

the body below the lesion. If the lesion involves both the gray and the white matter, there is a combination of the aforesaid symptoms. If the lesion is circumscribed, the symptoms that result will depend on the location of the lesion; for example, if the lesion is limited to a destruction of one-half of a segment, the Brown-Séquard syndrome occurs. In this case there will be atrophy, loss of reflexes, paralysis, and vasomotor and secretory disturbances in the distribution of the affected segment on the same side of the body. Below the lesion on the same side there will be spastic paralysis, increase of deep reflexes, and loss of muscle sense and partial loss of tactile sense. On the opposite side of the trunk below the lesion there will exist loss of pain and of temperature sense and partial loss of tactile sense.

In complete destructive lesions of the spinal cord there occur, below the lesion, flaccid paralysis, loss of all forms of sensation, abolition of all reflex activity, loss of bowel and bladder control and of muscle tone, marked trophic disturbances, etc.

No satisfactory explanation has ever been offered for the complete loss of all reflex activity and the abolition of muscle tone, etc., after complete lesions of the cord.

ANATOMY AND PHYSIOLOGY OF THE MEDULLA OBLONGATA, PONS, AND CRURA

The Medulla Oblongata.—The medulla is continuous with the upper end of the spinal cord. On its anterior surface is the anterior median fissure, which corresponds with that of the same name in the spinal cord, but it is separated from it by the decussating fibers of the pyramids. The masses of white matter lying on each side of the fissure, known as the *pyramids*, appear at first to be continuous with the anterior columns of the spinal cord, but in reality send only a small part of their fibers to the anterior column. The larger portion goes to the lateral column of the opposite side, to form the crossed pyramidal tract.

The pyramids are bounded laterally by the anterolateral fissure. From this fissure emerge the root-bundles of the hypo-

glossal nerve. Lateralward from these grooves, in the upper part of the medulla, are two oblong, egg-shaped elevations termed the *olives* (Fig. 22). Posterior to the olives is another groove, which corresponds with the posterolateral groove of the spinal cord. Posterior again to these grooves are two round elevations

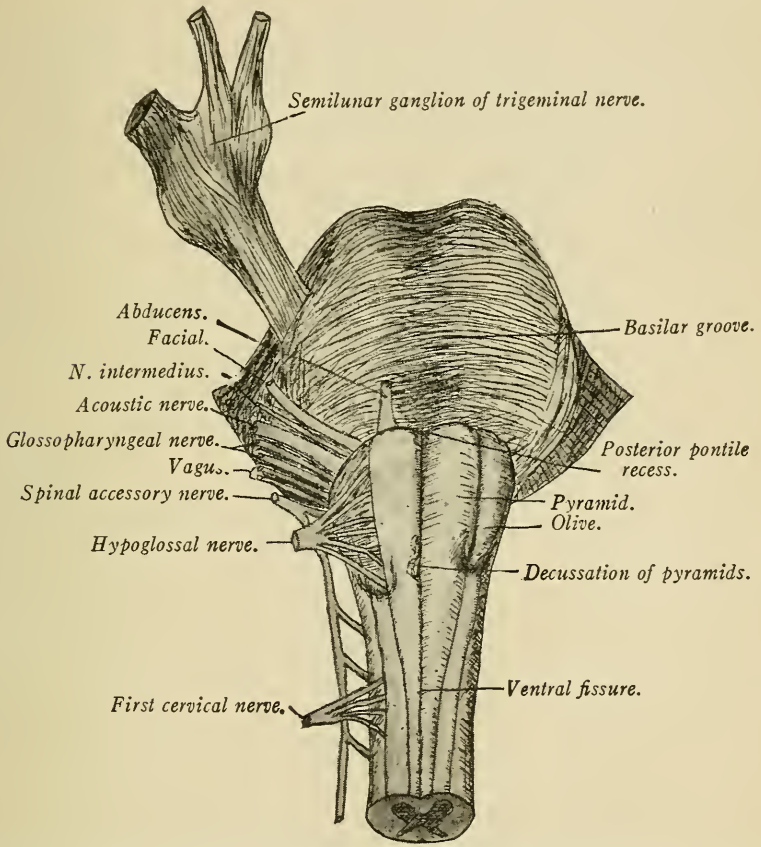


Fig. 22.—Anterior view of the pons and medulla, showing the superficial origin of certain cranial nerves. (Modified from Gray.)

called the *restiform bodies*, which are apparently continuous with the posterior column of the cord and disappear into the cerebellum. The posterior surface of the medulla is continuous with the posterior columns of the cord. The upper half of the posterior surface of the medulla is occupied by a triangular depression, the apex of the triangle being directed downward. This de-

pression is formed by the separation of the restiform bodies, and forms the lower half of the fourth ventricle (Fig. 23).

The medulla is covered by the pia and arachnoid, which cover it loosely, forming a large space between its internal surface and the pia mater. This space is particularly well marked on the posterior aspect of the medulla.

The pia sends septa into the medulla, and is reflected on its posterior aspect, between it and the cerebellum, where it is covered by epithelial cells, forming the inferior tela choroidea. At the lower edge of the tela choroidea, just above the calamus scriptorius, is a small opening in the pia through which the intraventricular space connects with the subdural space. This opening is called the *foramen of Magendie.* On each side of the medulla, in the angle between it and the cerebellum, are two other small openings in the pia, which also allow communication between the intraventricular space and the subdural space. These openings are called the *foramina of Retzius.*

The medulla presents the following important structures for consideration: On the anterior aspect we see, on either side of the anterior median fissure, a round, pyramidal elevation that extends from immediately beneath the pons to the lower end of the medulla; these are called the *pyramids of the medulla.* External to the pyramid, in the anterolateral groove, appear a number of nerve-fibers. These fibers form the twelfth or hypoglossal nerve, and the point of their appearance is called the superficial origin of this nerve. External to the anterolateral groove, immediately beneath the pons, is an oblong, egg-shaped elevation known as the olive. The olivary bodies in the lower portion are frequently crossed by arched fibers called the *external arcuate fibers.* On each side of the central line, immediately below the pons, and almost in the groove between the pyramids and the olivary body, fibers appear that form the abducens or sixth nerve. The point of appearance of these fibers is called the superficial origin of the sixth nerve.

In the posterolateral groove numbers of fibers appear that mark the superficial origin of the following nerves: Beginning above, immediately beneath the pons, are the seventh or facial, the eight or auditory, the ninth or glossopharyngeal, the tenth

or pneumogastric, and the accessory portion of the eleventh or spinal accessory nerve (Fig. 22).

On the posterolateral aspect of the medulla are two rounded elevations that disappear into the cerebellum. They are called the restiform bodies, or inferior peduncles of the cerebellum.

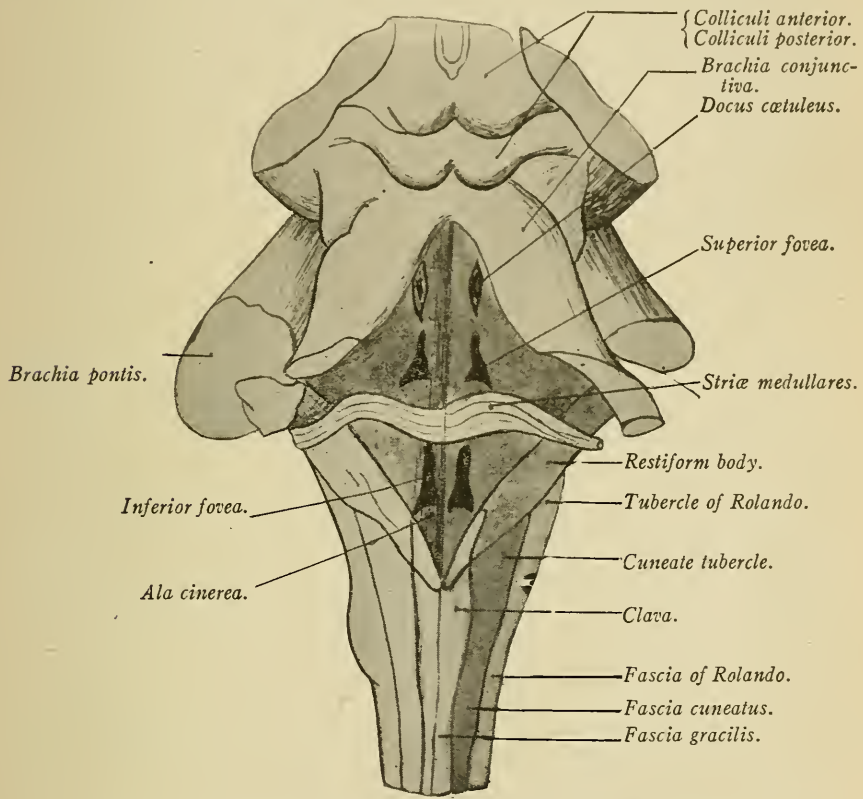


Fig. 23.—Posterior view of pons and medulla (Whitaker).

The dorsal surface is divided into halves by the posterior median fissure, continued upward from the cord. On the dorsal surface of the medulla, in each half, are four columns or tracts, separated from one another by slight grooves. Close to the mid-line are the fasciculus gracilis and fasciculus cuneatus; external to these is the fasciculus of Rolando, and external to this is the direct cerebellar tract. The upper half of the dorsal surface of the medulla is somewhat wider than the lower half, the increase

in width being progressive from below upward. The lower portion is the closed portion of the medulla; the upper portion forms an open triangle, which is the lower half of the fourth ventricle. On the posterior surface of the medulla are slight elevations that contain the nucleus cuneatus and the nucleus gracilis (Fig. 23).

Pons Varolii.—The pons presents the appearance of an inverted bridge that seems to span the medulla. It is a broad, transverse, striated band that disappears laterally into the cerebellum. The lateral portion of the pons, where it disappears into the cerebellum, are called the middle peduncles of the cerebellum. Upon the lateral aspect of the pons, piercing its transverse fibers, are a number of nerve-fibers that go to form the fifth or trigeminal nerve. The point at which they appear is called the superficial origin of the fifth.

The posterior aspect of the pons is occupied by the upper half of the fourth ventricle (Fig. 23).

The Fourth Ventricle.—This cavity contains a small amount of fluid. It is continuous below with the central canal of the spinal cord, going above directly into the aquæductus cerebri. The anterior wall is formed by the posterior aspect of the upper half of the medulla and the pons (Fig. 23).

This ventricle is a lozenge-shaped fossa, the lower part, between the restiform bodies, belonging to the medulla oblongata, the medial part to the pons Varolii, and the upper part to the isthmus. The lower portion is approximately triangular. It appears much deepened below, and is marked by several furrows that converge downward. The peculiar convexity of its surface in the lower part has given to it the name *calamus scriptorius*. Along its lateral and inferior margin is attached the tela choroidea. The upper portion is bounded on each side by the superior cerebellar peduncles, and is covered by the anterior medullary velum. It gradually narrows into the aqueduct of Sylvius. The fourth ventricle is divided into symmetric halves by a median fissure. Close to this fissure or sulcus on each side is a ridge, and external to this, in the upper part of the floor of the fourth ventricle, is a dark-bluish area called the *locus cæruleus*. In the inferior part this ridge gives off a triangular area, called the *trigonum hypoglossi*. External to the trigonum hypoglossi

is a narrow area called the *ala cinereia*. Running transversely

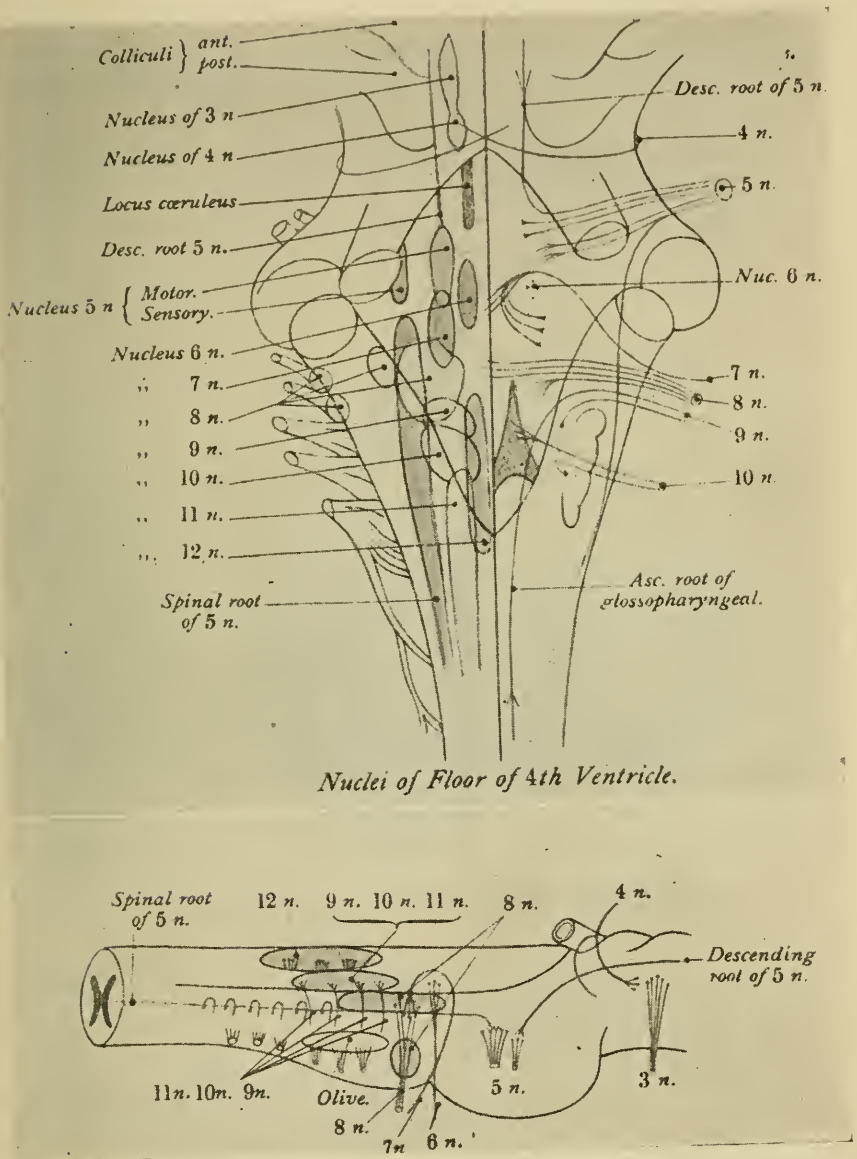


Fig. 24.—Nuclei of the floor of the fourth ventricle. (Whitaker, "Anatomy of the Brain and spinal Cord," after Edinger.)

across the fourth ventricle, about its median part, are superficial white fibers—the medullary striæ or striæ acousticæ—which

arise from elevations at the lateral angle of the fourth ventricle, called the acoustic tubercles. These fibers disappear in the depths of the median fissure, or even before going that far.

The roof of the fourth ventricle—the anterior portion—is formed by the cerebellum and the superior cerebellar peduncles. The latter are connected by a lamina of white matter, the anterior medullary velum or valve of Vieussens, which is continuous with the white substance of the cerebellum. The lower part is covered by the posterior medullary velum and the tela choroidea inferior, which is a double fold of the pia mater. From its inner

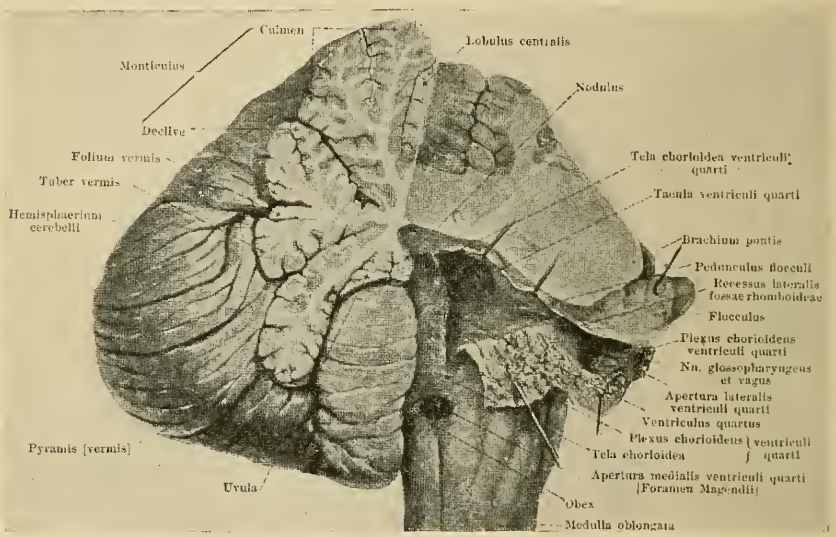


Fig. 25.—The roof of the fourth ventricle (Spalteholz).

surface are given off a number of projections containing blood-vessels; these form what is known as the choroid plexus of the fourth ventricle (Fig. 25).

The *aquæductus cerebri*, or aqueduct of Sylvius, is a narrow canal, about 5 cm. long, which passes from the upper end of the fourth ventricle through the crura cerebri to the third ventricle. In cross-section it is **T** shaped.

Cerebral Peduncles.—These are two large, striped, white columns, situated close together. They emerge from beneath the upper margin of the pons, diverge from each other, and pass upward and lateralward, disappearing beneath the optic tracts

into the cerebrum. The ventral and lateral surfaces are free, and are called the *basis pedunculi.* That portion of the peduncles posterior to this is called the tegmentum. Upon the dorsal aspect of the cerebral peduncles are the corpora quadrigemina, which are four rounded, arched elevations—two superior and two inferior. Below the corpora quadrigemina, and extending over the fourth ventricle as part of its roof, are the brachia conjunctivæ cerebelli, or superior cerebellar peduncles (Fig. 26).

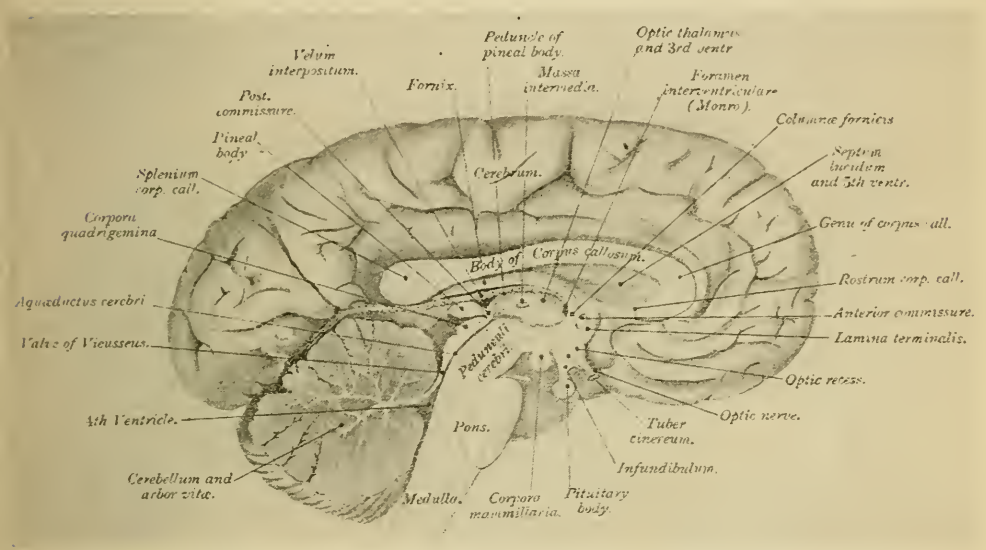


Fig. 26.—Mesial section of brain, showing the fourth ventricle, aqueduct of Sylvius; cerebral peduncles, corpora quadrigemina, pineal body, etc. (Whitaker, "Anatomy of the Brain and Spinal Cord.")

Corpora Quadrigemina.—These are situated upon the dorsal aspect of the crura cerebri, immediately in front of and directly in contact with the superior cerebellar peduncles. They are subdivided by a median flat and by a transverse groove into four whitish, hemispheric elevations; thus we have an inferior and a superior corpus quadrigeminus on each side. The superior or anterior corpora quadrigemina are larger than the posterior or inferior. In the median groove, between the two superior corpora quadrigemina, lies the pineal gland. Each corpus quadrigeminus is continued lateralward and upward by the corpora quadrigeminal arm. The superior corpora quadri-

gemina are connected by means of these arms with the external geniculate bodies, and the inferior corpora quadrigemina connect by means of their arms with the internal geniculate body.

STRUCTURE OF THE MEDULLA, PONS, AND CEREBRAL PEDUNCLES

On transverse section of the lower part of the medulla it will be seen that its gray matter presents an arrangement very

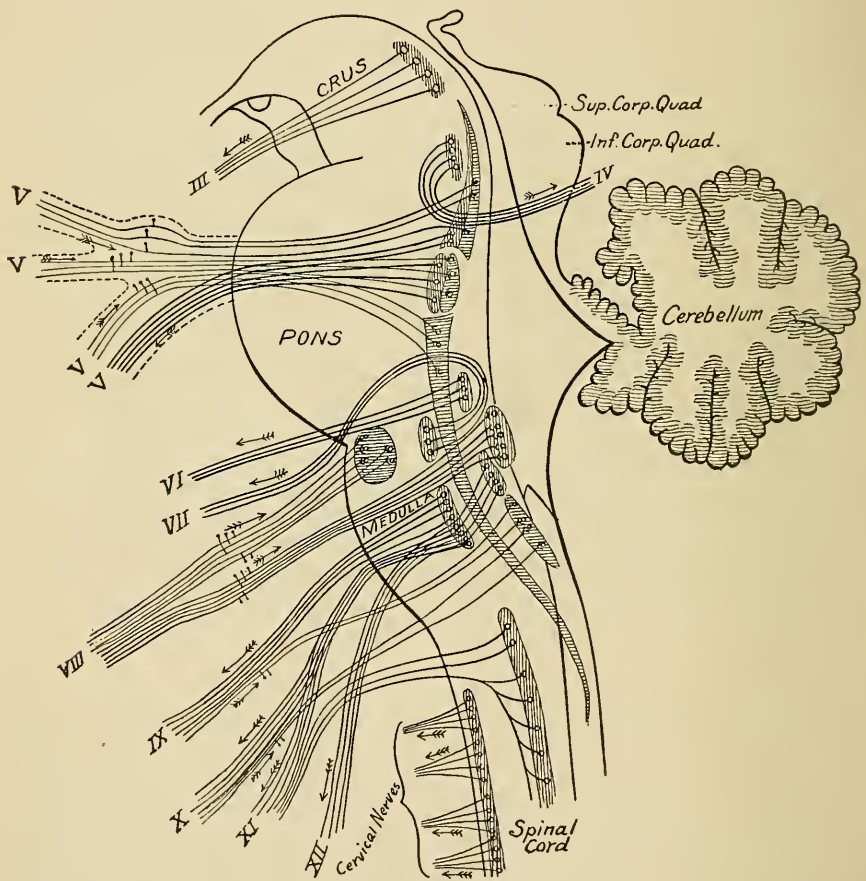


Fig. 27.—Sagittal longitudinal section (medulla, pons, and crura). Diagrammatic.

similar to that of the gray matter of the spinal cord. Higher up, however, the arrangement of the gray matter becomes

much changed. The anterior horns lose their characteristic shape, owing to the fact that the fibers of the pyramidal tracts, in their course from the medulla to the lateral columns of the cord, cut their way through the neck of the anterior horns; thus detached, the head of the horn enlarges and is pushed to the lateral aspect of the medulla (Fig. 28).

The central canal of the spinal cord opens out into the floor of the fourth ventricle, and the base of the anterior horns appear on the ventricular floor as a narrow gray tract. The greater part of the anterior horn is replaced by a network of cells and fibers. This network constitutes the anterior part of the *for-*

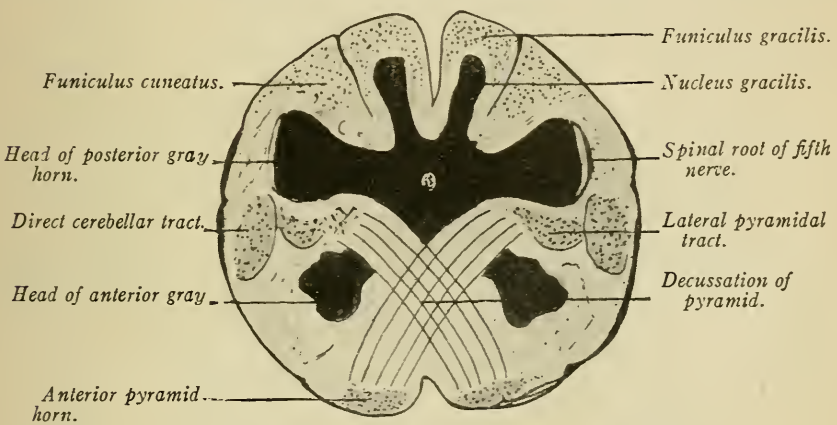


Fig. 28.—Transverse section through medulla, showing motor decussation.

matio reticularis. On reaching the medulla the gray matter of the posterior horn also takes up a lateral position. The gray matter at the base of the posterior horn increases in amount, and forms the nucleus of the column of Burdach and that of the column of Goll. Around the cells in these nuclei the fibers of the columns of Goll and Burdach terminate, and from these cells arise other fibers that cross the middle line, forming the sensory decussation (Fig. 29). Then, turning upward, they go to form the lemniscus, or fillet, which is one of the pathways for the conduction of sensation. The olivary nucleus is contained within the olivary body, which must be divided in order to bring it into view. It consists of flask-shaped, multipolar nerve-cells,

arranged concavely, with an opening on its inner aspect. Through this opening, called the hilum, enters the olivary

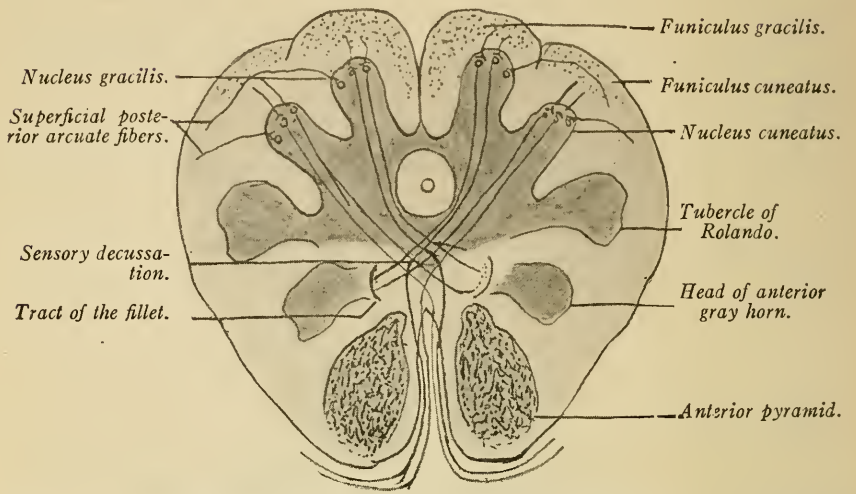


Fig. 29.—Transverse section through medulla, showing sensory decussation.

peduncle, which connects the olivary body with the corpus dentatum situated in the opposite side of the cerebellum.

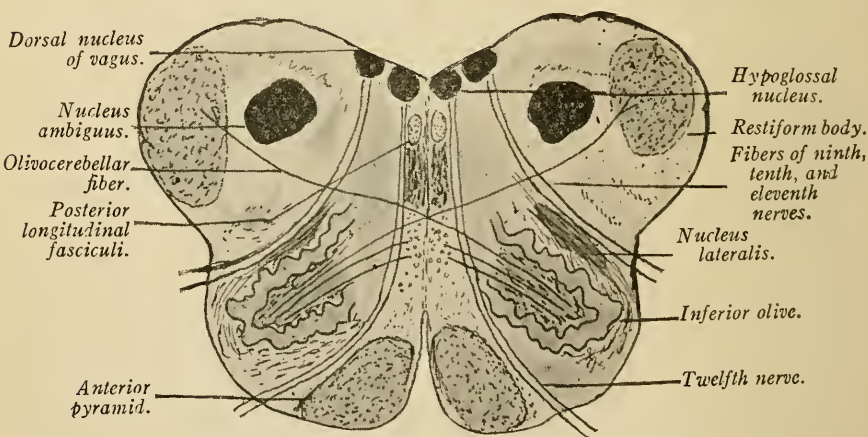


Fig. 30.—Transverse section of medulla, cutting olivary nucleus.

These fibers cross through the reticular formation, and form the principal part of the inferior cerebellar peduncle (Fig. 30).

In the pons the fibers of the motor pathway are arranged in bundles, being interlaced with the transverse fibers (Fig. 31). Behind the pyramids are two smaller columns, the corticopontine and the caudate cerebellar, both of which connect the cerebrum with the pontine nuclei, and thence, through the brachia pontis, with the cerebellum. Posterior to these fibers, near the dorsal aspect of the pons, are the following distinct set of longitudinal fibers: First, the lemniscus or fillet, near the midline, in the ventral part of the formatio reticularis. This is the continuation

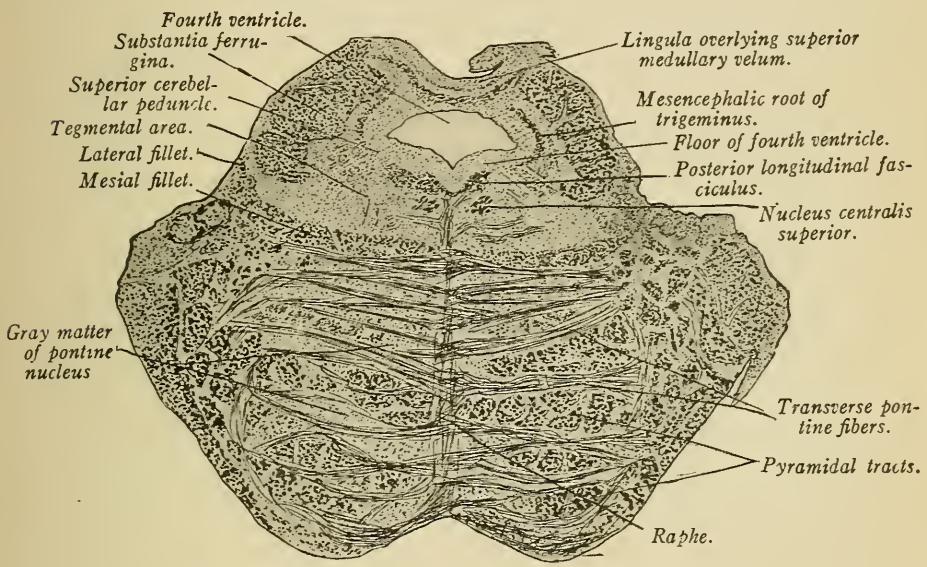


Fig. 31.—Transverse section of pons at upper part.

of the sensory tracts from the nuclei of Goll and Burdach of the medulla. In the lateral part of the formatio reticularis of the pons are found the fibers of the anterolateral tract, which have ascended from the spinal cord through the lateral columns and the formatio reticularis of the medulla. This set of fibers pursues two courses: the one set, called the spinothalamic, form a direct route for the transmission of the sensations of touch, pain, and temperature; the other, the ventro-spinocerebellar, constitute an indirect route through the cerebellum for the transmission of these same types of sensation.

Cross-section of the cerebral peduncles will show that each consists of two portions—a posterior, called the *tegmentum*, and an anterior, called the *crusta*. These two parts are separated from each other by a narrow stratum of gray matter called the *substantia nigra* (Fig. 32). The **tegmentum** is a larger division of the crus, and is composed of a reticulum of longitudinal and transverse fibers interspersed with gray matter. The gray matter consists of the reticular formation continuous with that of the pons and the medúlla. In this, in addition to many scattered nerve-cells are found two definite collections of gray matter—one around the aqueduct of Sylvius and the other known as the red nucleus. The red nucleus lies beneath the anterior pair of cor-

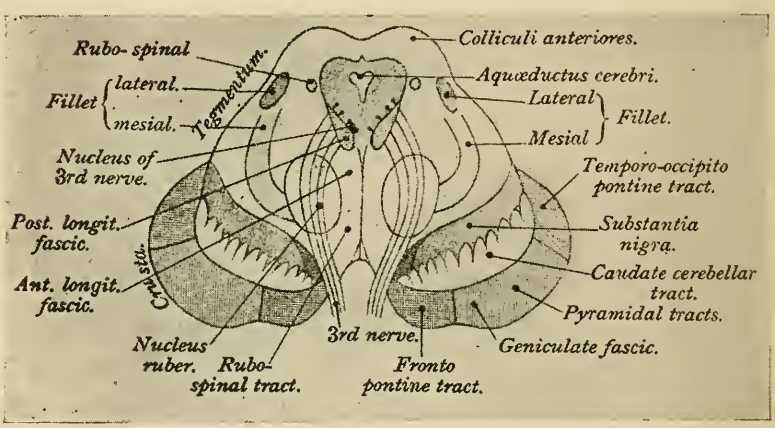


Fig. 32.—Transverse section of the cerebral peduncles (Whitaker).

pora quadrigemina, and consists of multipolar nerve-cells. It is the primary termination of the superior cerebellar peduncles. The gray matter around the aqueduct of Sylvius gives origin to the third and fourth cranial nerves. The white matter of the tegmentum consists of longitudinal and transverse fibers. The chief strands are: First, the *superior cerebellar peduncles*, which connect the cerebellum with the opposite red nucleus, and through this, probably, with the cerebral cortex. Second, the *fillet*, which is the continuation upward of the main sensory pathway from the medulla and pons. This, when continued upward, is found to terminate around the cells in the optic thalami, cerebral cortex, and inferior and superior corpora

quadrigemina. Third, the *rubrospinal tract*, which consists of the fibers originating in the cells of the red nucleus, crossing the opposite side, and descending through the medulla to the lateral column of the spinal cord. Its fibers terminate around the cells in the anterior horn.

The transverse fibers of the crura are the fibers of the third and fourth cranial nerves on their way from their deep to their superficial origin. The **crusta,** or base of the peduncles, lies in front of substantia nigra, and is composed of the following tracts: The internal one-fifth is occupied by the frontopontine tract, which is made up of fibers that connect the frontal lobe of the cerebrum with the pontine nuclei. The middle three-fifths are taken up with the pyramidal fibers, in the following order: First appear those fibers that transmit impulses from the lower precentral convolution; that is, those that transmit stimuli to the nuclei of the cranial nerves. External to these are those fibers that originate somewhat higher in the precentral convolution, and transmit impulses that effect the movements of the upper extremities. External to these are the fibers that transmit impulses that bring about the movements of the trunk and the lower extremities. The external one-fifth is occupied by the temporo-occipitopontine tract.

Functions of the Medulla, Pons, and Crura.—These structures contain fiber-tracts that conduct stimuli upward and downward. Those situated anteriorly are the motor pathways that transmit impulses from the brain, which bring about voluntary movements and which inhibit activities not desired. Posterior to these pathways are the tracts through which ascend the stimuli that, on being impressed on consciousness, we perceive as the various types of sensation. Interspersed between these two pathways, at various levels, are the cranial nerves. The relative spatial relationship of the nuclei and the fibers of these nerves to the ascending and descending pathways can best be understood from a study of longitudinal and cross-sections of these structures (Figs. 24, 27, and 33).

Corpora Quadrigemina.—The superior pair of corpora quadrigemina consists of several layers of gray and white matter. The posterior pair is composed of a central gray core and a white

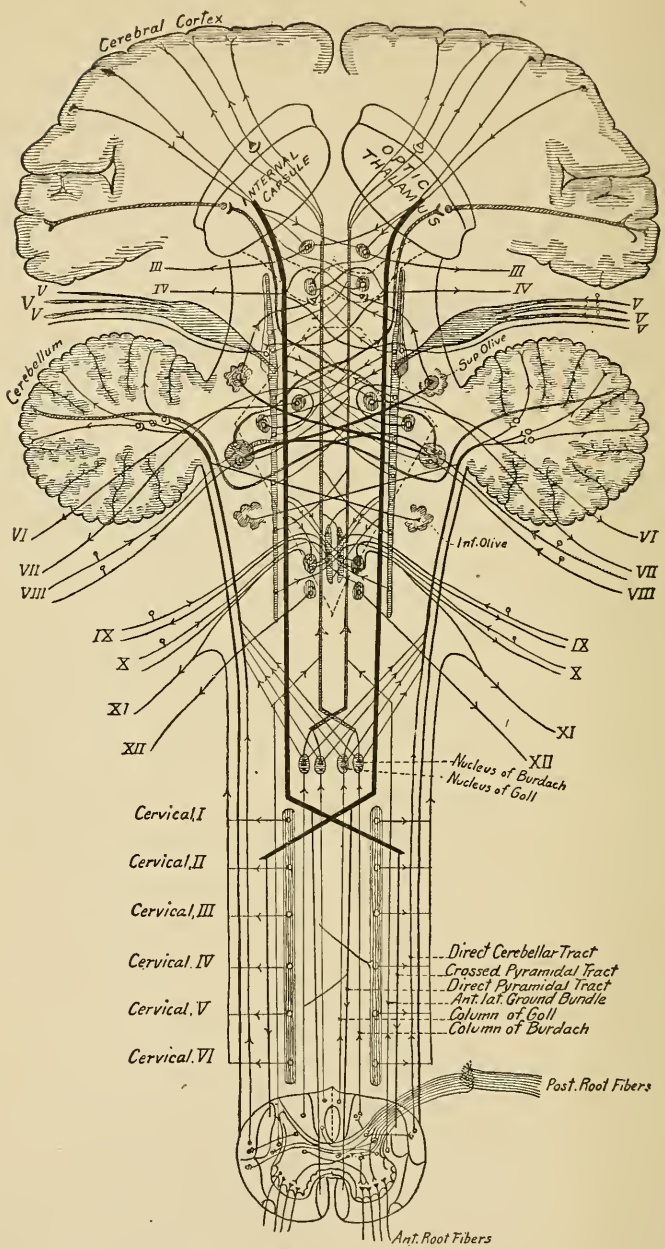


Fig. 33.—Longitudinal diagrammatic section of the crura, pons, medulla, and upper part of spinal cord, showing the principal ascending and descending tracts, the nuclei and fibers of the cranial nerves from the third to the twelfth, and their relation to each other.

cortex. The superior corpora quadrigemina and the external corpora geniculata are concerned with vision; the inferior corpora and the internal corpora geniculata are concerned with hearing. These bodies vary in size in different animals, and bear a direct relation to the function of sight and to the acuteness of hearing.

THE CEREBELLUM

The cerebellum, or small brain, is situated behind the pons and medulla, and is contained within the occipital fossa. It lies beneath the posterior lobes of the cerebrum, from which it is

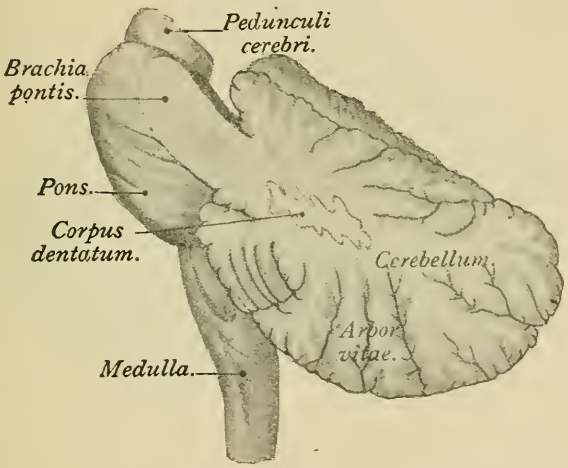


Fig. 34.—Median section of the cerebellum. (Whitaker, "Anatomy of the Brain and Spinal Cord.")

separated by the *tentorium cerebelli*. It measures about three to four inches transversely, and from two to two and one-half inches anteroposteriorly. It is about two inches thick in the center, becoming gradually thinner as the outer circumference is approached. The surface of the cerebellum is traversed by numerous narrow furrows that vary in depth (Fig. 34).

In structure the cerebellum consists of gray and of white matter, the former being darker than that of the cerebrum, and occupying the surface; the latter occupies the interior. It

consists of a median division, called the vermiform process; two lateral divisions, the cerebellar hemispheres; and three peduncles —the superior, middle, and inferior—by means of which the organ is brought into communication with the cerebrum, the pons, and the medulla.

The anterior margin of its surface presents in the middle a flat notch, the *anterior cerebellar incision*, which lodges the pons and the medulla. The cerebellar hemisphere presents three surfaces—an anterior, a superior, and an inferior. The superior and inferior are separated from each other by the great horizontal

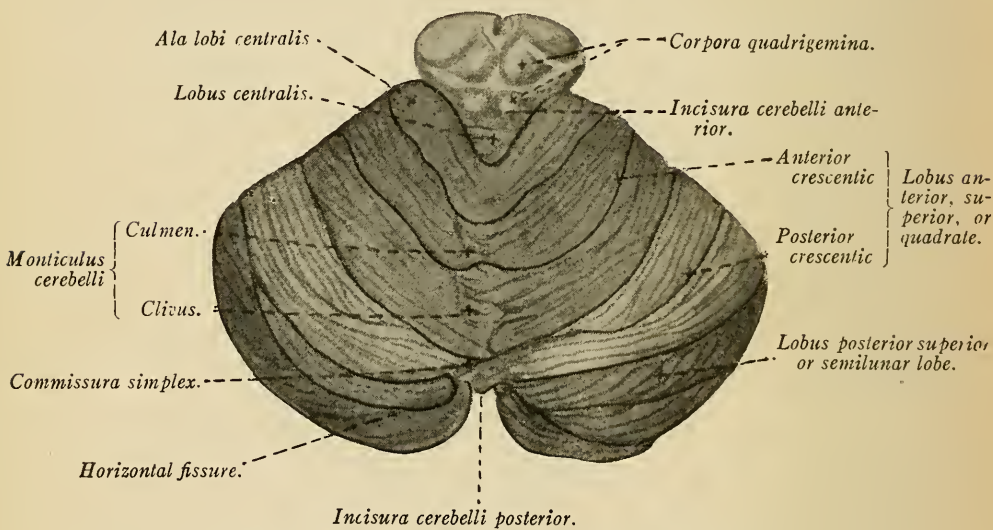


Fig. 35.—Upper surface, cerebellum (Whitaker).

fissure. The cerebellar hemisphere consists of a central white core, a little central gray matter, and, on the surface, numerous crescentic gray laminæ with their convexity directed backward.

The upper surface of each cerebellar hemisphere is concave, and they are separated from each other in the midline by a longitudinal elevation which is the upper surface of the vermis cerebelli (Fig. 35).

The under surface of each hemisphere is convex, and the two hemispheres are divided from each other by a median furrow in which is seen the inferior surface of the vermis cerebelli (Fig. 36).

Posteriorly, the hemispheres are separated by the *posterior cerebellar groove*, which receives the free anterior margin of the falx cerebelli.

The anterior surface of the cerebellum presents in the midline a notch, the *anterior cerebellar groove*, which lodges the pons Varolii and the medulla. When these structures are removed, there are revealed in the midline the following structures: The anterior end of the vermis cerebelli, the valve of Vieussens, and the cavity of the fourth ventricle; on each side is seen a cerebellar hemisphere with its three peduncles; externally, in the midline, is the longitudinal sulcus, in which is lodged the flocculus.

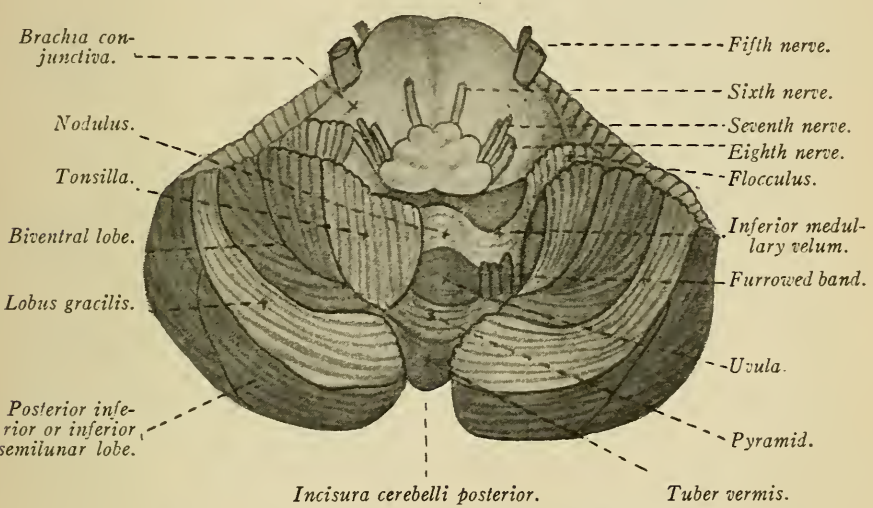


Fig. 36.—Under surface of cerebellum (Whitaker).

The surfaces of the cerebellar hemispheres present a laminated appearance. The furrows, extending continuously on the upper surface over the vermis, run approximately parallel to the posterior margin. The superior and inferior surfaces of the cerebellum are divided into lobes, some of which are very poorly defined.

The Peduncles of the Cerebellum.—These are bilateral bands of white matter, three to each hemisphere—the superior, the middle, and the inferior. They connect the cerebellum with the cerebrum, with the pons, and with the medulla (Fig. 37).

The *superior peduncles*, or *brachia conjunctiva*, are two white bands situated beneath the anterior part of the cerebellum. They connect the white substance of the hemispheres with the cerebral peduncles. They run upward and forward, forming part of the lateral walls of the upper part of the fourth ventricle, the interval between them being bridged over by a valve of Vieussens or the superior medullary velum. As they ascend

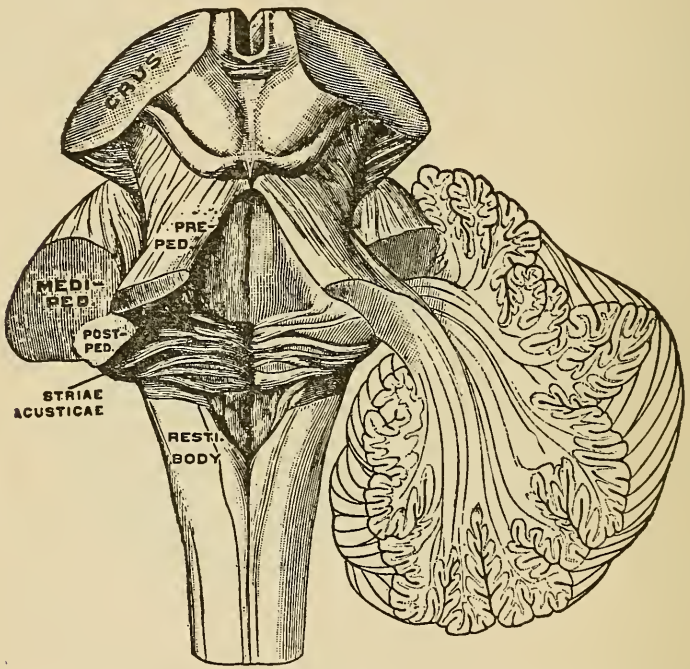


Fig. 37.—The peduncles of the cerebellum. On the left the three peduncles have been cut at their entrance into the cerebellum; on the right side they are shown penetrating the cerebellar hemisphere (Poirier and Charpy).

the peduncles meet in the midline and form part of the roof of the fourth ventricle. The fibers that form the superior peduncles originate chiefly in the corpus dentatum, which is a mass of gray matter in the interior of the cerebellum. Some of the fibers of these peduncles also originate in the cells of the cerebellar cortex. The majority of the fibers of the peduncles decussate with those of the opposite peduncle, and then pass upward as a distinct bundle with the uncrossed fibers. They

then divide into the ascending and descending branches. The ascending branches terminate around the cells in the cerebrum, cells in the red nucleus, in the optic thalamus, etc. The descending branches pass downward in the reticular formation of the pons and medulla, ending around the nuclei of the motor cranial nerves.

The *middle peduncles*, or *brachia pontis*, are a continuation of the lateral part of the pons into the cerebellum, and are made up of the fibers that lie above the superficial origin of the fifth cranial nerve. The fibers of the middle peduncles become incorporated with the pons, forming deep and superficial transverse fibers. Some of the fibers of these peduncles are commissural, connecting the two sides of the cerebellum; others terminate around the pontine nuclei of the opposite side, and a few terminate in the same side (Fig. 38).

The *inferior cerebellar peduncles*, or *restiform bodies*, are two round elevations that ascend from the medulla to enter the white matter of the cerebellar hemispheres. They form the lateral boundaries of the lower part of the fourth ventricle. The fibers of the inferior peduncles originate in the spinal cord, in the medulla, in the olivary bodies, and in the cerebellum. Spinal fibers; these are the direct cerebellar tract ascending from the same side of the cord, and the arciform fibers which have originated in the nuclei of the posterior column of the same and opposite side; the fibers from the medulla originate in the sensory portion of the nuclei of the tenth, ninth, fifth, and the vestibular portion of the eighth nerves; the olivary fibers originate in the olives of the opposite side; the cerebellar fibers are descending fibers from the cerebellum to the nuclei of the cranial nerves and to cells in the spinal cord. Most of the fibers of the inferior cerebellar peduncles end around or originate in the cells of the vermis of the opposite side.

The *valve of Vieussens*, the *superior medullary velum*, is a delicate sheath of nerve substance, triangular in shape, the apex being directed forward. It forms part of the roof of the upper division of the fourth ventricle, and is placed anteriorly across the interval left between the two superior cerebellar peduncles. The gray and white matter of this structure is continuous with the gray and white matter of the cerebellar hemispheres.

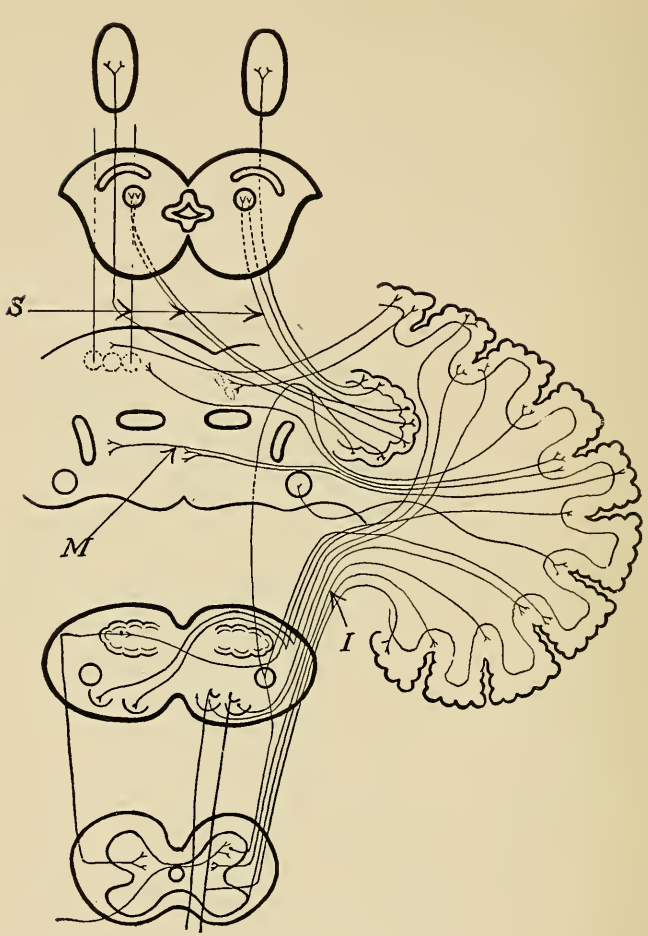


Fig. 38.—Diagram showing course of fibers which make up the cerebellar peduncle: *S*, Superior cerebellar peduncles, made up as follows: fibers originating in the cells of the red nucleus in the cerebellar peduncles of the same and opposite sides; fibers originating in or terminating around cells in the corpora quadrigemina. *M*, Middle cerebellar peduncle, made up as follows: fibers which originate in the pontile nuclei of the same and opposite side, commissural fibers which connect the two cerebellar hemispheres with each other and with the middle lobe of the cerebellum. *I*, Inferior cerebellar peduncle, made up as follows: direct cerebellar tract which originates in the cells of Clarke's column; external and internal arciform fibers which originate in the cells in the nuclei of Goll and Burdach on the same and opposite side; fibers which originate in the dentate nucleus of the opposite inferior olive; fibers from the nuclei of the cranial nerves, especially the vestibular portion of the eighth nerve.

Structure of the Cerebellum.—The cerebellum is made up of gray and white matter. The gray matter consists of two parts: that on the surface is called the cortex cerebelli, and that in its interior is known as the nuclei cerebelli. The cortex cerebelli forms a thin lamina that is folded upon itself in a series of leaves or blades; these, upon section, present, with the white matter, a tree-like appearance called the arbor vitæ.

The nuclei cerebelli are situated in the cerebellar hemisphere—corpora dentata—one in each hemisphere. They are similar in structure to the corpora dentata of the olivary body, and are closely connected to it. They consist of brown nerve substance inclosing white matter, the whole forming a pouch-like structure that is open at its upper and inner sides. Through these openings nerve-fibers enter the corpus dentatum. These fibers can be traced from the superior peduncles, from the valve of Vieussens, and from the inferior cerebellar peduncles. There are three other nuclei within the cerebellum. These are situated near the midline, internal to the corpus dentatum, and are named the nucleus globosus, the nucleus emboliformis, and the nucleus fastigii.

The white matter of the cerebellum is made up of three sets of fibers—projection, commissural, and association. The projection fibers constitute the peduncles of the cerebellum; the commissural fibers extend from one hemisphere to the other, while the association fibers connect the various convolutions with one another.

Microscopic Structure of the Cerebellum.—The gray cortex of the cerebellum consists of three layers—the outer or molecular layer; a middle layer of Purkinje's cells, and an inner or granular layer. The outer layer forms a clear gray stratum on the surface of the cerebellum. It is composed of neuroglia and of large and small molecular cells. The latter are multipolar cells, their axis-cylinder processes running transversely into the granular layer, and giving off at right angles branches whose terminal twigs envelop the bodies of the cells of Purkinje. Owing to this arrangement, many of the cells of this layer are called " basket-cells" (Fig. 39).

The middle layer is formed of the *cells of Purkinje*. These

cells are characteristic of the gray matter of the cerebellum, and, owing to their shape, have also been called "antler cells." The large, flask-shaped cell-body and the dendritic process are directed toward the surface of the cerebellum. They divide and subdivide like the branches of a tree or the horns of a deer, They are closely surrounded by many fine fibrilla. From the

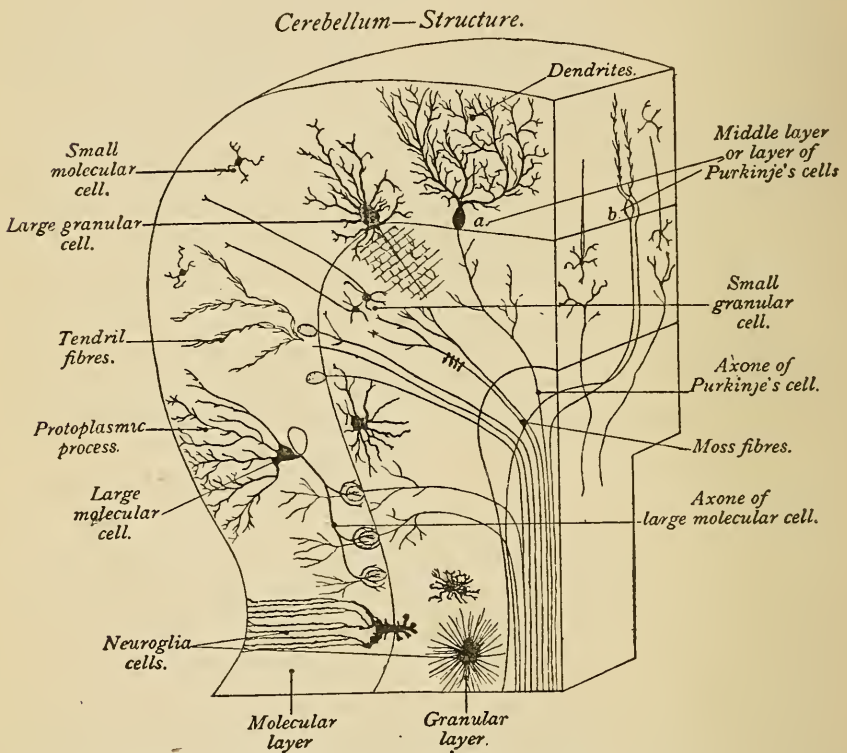


Fig. 39.—Microscopic structure of cerebellum. Diagrammmatic. (Whitaker, "Anatomy of the Brain and Spinal Cord.")

larger end of the cell a single, slender axis-cylinder process is given off. This sends off a lateral process and then enters the white matter.

The inner layer consists of large and small granular cells, neuroglia, a fine network of fine interlacing fibrilla of axis-cylinder processes passing toward the molecular layer. Fibers, known as "moss fibers," are also present; they ascend from the

gray matter of the cord, and end as short, thick branches or as little mossy tufts in the cortex. Fibers that end in a plexus in the molecular layer, and are called "climbing" or "tendril fibers," are also seen.

Functions of the Cerebellum.—The hemispheres of the cerebellum may be even irritated or removed without giving rise to any symptoms. These structures are probably connected with the sympathetic system and with the frontal lobes, and exert an influence upon the emotions.

The middle lobe, or vermis, seems to be that portion concerned mostly with the important activities of the cerebellum. It has a marked influence on the muscle movements; for example, disturbance of the middle lobe may cause forced movements, tremors, and cataleptic attitudes. The cerebellum is the controlling center wherein are combined the many stimuli that result in coördination. Diseases of the cerebellum may bring about incoördination; abnormalities of movements; ataxia; vertigo; speech disturbance; nystagmus; adiadokokinesis; disturbance of equilibrium, etc.

THE CEREBRUM

The cerebrum is by far the largest division of the brain. It presents a surface convex from before backward and from side to side, which corresponds to the inner surface of the skull. Its inferior surface occupies the anterior and middle fossæ of the skull, and posteriorly rests upon the upper surface of the tentorium cerebelli, which separates it from the cerebellum.

It is an egg-shaped mass, larger behind than in front, and composed of two symmetric halves that are partly united in the midline. Each half presents a flat median surface, a convex lateral surface, and an irregular inferior surface. These halves, or, as they are called, cerebral hemispheres, are united by a thick band of white matter known as the corpus callosum. They are separated from each other by the great longitudinal fissure, in which is placed the falx cerebri. Each hemisphere is composed of a white stalk or peduncle, surrounded by a convoluted gray

mass. This is traversed by indentations called fissures, or sulci, which divide the mass into larger and smaller folds, which are termed convolutions, or gyri. Internally, the hemispheres consist of masses of gray matter,—the subcortical ganglia,—of white matter, made up of nerve-fibers, and of a cavity,—the cerebral ventricle.

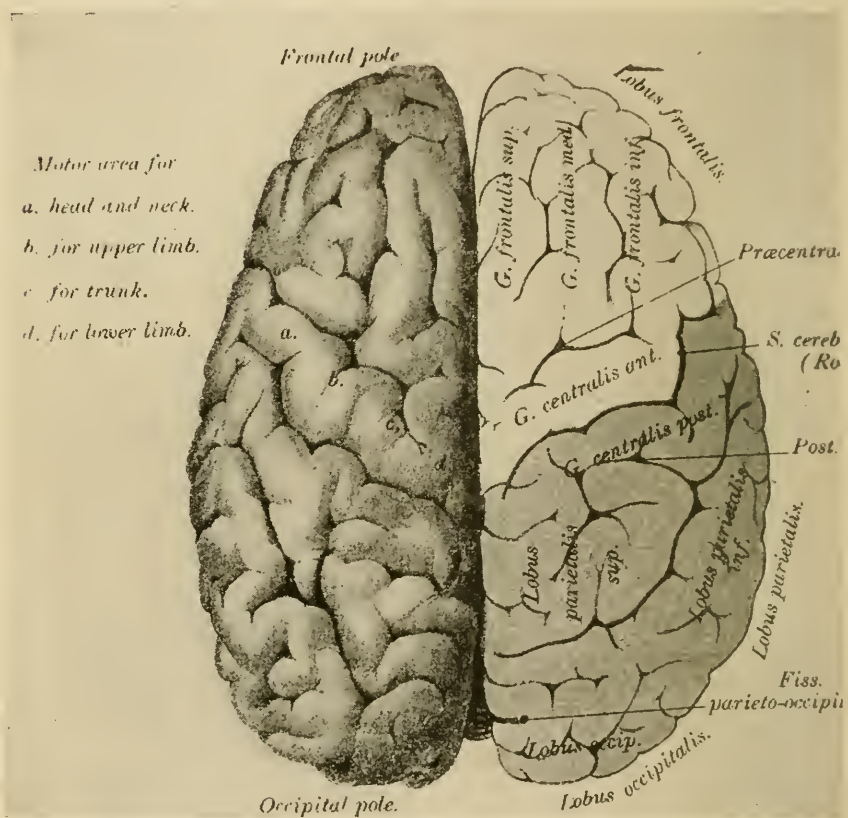


Fig. 40.—Cerebrum as seen from above (Whitaker).

Fissures.—The surface of a cerebral hemisphere is marked by numerous elevations and depressions, some of which are practically constant as to location. The largest of these depressions divide the surface of the cerebrum into lobes, and the smaller ones subdivide these lobes into convolutions or gyri. The most constant and most evident fissures are the fissure of Sylvius, the fissure of Rolando, and the parieto-occipital fissure.

The *fissure of Sylvius* is a deep indentation which begins near the midline, on the under surface of the hemisphere, at a point called the anterior perforated space. It runs transversely upward and outward on the base of the hemisphere to its lateral aspect, where it divides into two limbs—an anterior short vertical ascending limb, and a posterior one, which runs horizontally backward on the surface of the hemisphere, and is therefore called the horizontal limb.

The *fissure of Rolando* is one of the first fissures to appear in the development of the brain. It commences above, at the

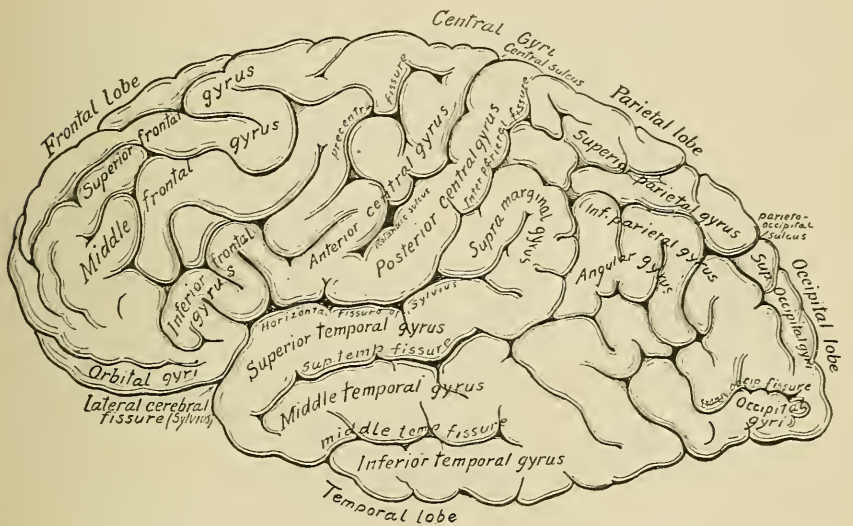


Fig. 41.—Lateral view of brain, showing lobes, fissures, and convolutions.

median longitudinal fissure, in many cases on the median surface of the hemisphere, posterior to the anterior half of the hemisphere. It descends obliquely forward across the outer surface of the cerebrum, and ends below, near the anterior part of the horizontal limb of the fissure of Sylvius, which it rarely enters.

The *parieto-occipital fissure* appears on both the outer and the median aspect of the hemisphere. Externally it is a short cleft on the outer aspect of the hemisphere, at about the junction of the anterior five-sixths and the posterior sixth of the total distance from the posterior to the anterior pole. The internal

portion of this fissure is well marked and descends vertically in the posterior portion of the median aspect of the hemisphere (Fig. 43).

These fissures divide each cerebral hemisphere into four principal lobes, which take their names from the portion of the skull beneath which they lie. These are the frontal, the parietal, the temporal, and the occipital lobes. The fifth lobe, which is called the *central lobe*, or the *island of Reil* (Fig. 42), is hidden within the fissure of Sylvius. Two other lobes are sometimes described: the olfactory and the limbic lobe.

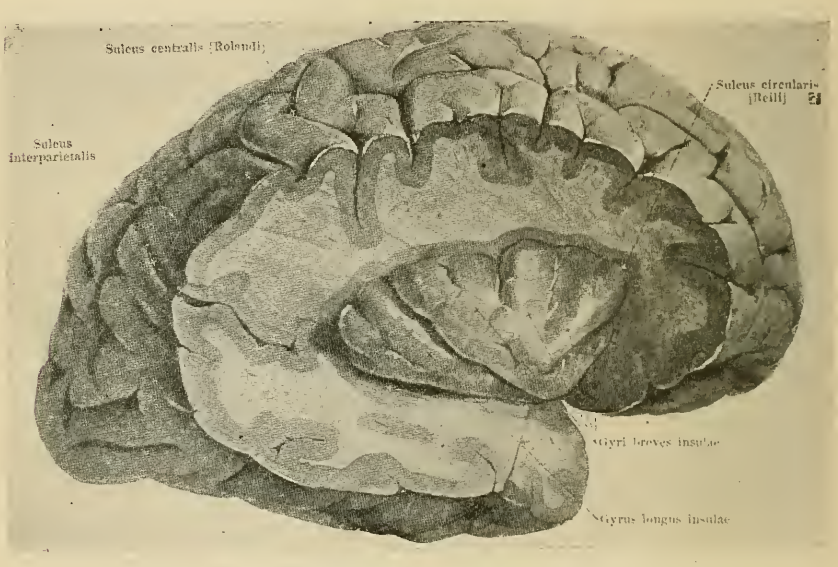


Fig. 42.—Central lobe, or island of Reil (Spalteholz).

Lobes of the Brain.—The **frontal lobe** is pyramidal in shape, occupying the anterior portion of the cerebral hemisphere. It is limited posteriorly by the fissure of Rolando, and inferiorly by the fissure of Sylvius. It is separated from the frontal lobe of the opposite side by the great longitudinal fissure. It has an outer, inner, and an inferior surface. The outer surface is marked by three important sulci—the precentral, the superior and the inferior frontal sulci, which divide this portion of the frontal lobe into four important convolutions: the precentral or ascending frontal convolution, which is immediately in front of

the fissure of Rolando, the superior, the median, and the inferior frontal convolutions.

The posterior part of the left inferior frontal convolution, surrounding the ascending limb of the fissure of Sylvius, is called Broca's convolution. In left-handed individuals this name is applied to a similar portion of the brain situated on the right side. The inferior or orbital surface of the frontal lobe is limited posteriorly by the fissure of Sylvius, and internally by the great longitudinal fissure. It is divided by the sulcus orbitalis into three convolutions. These are prolongations of the convolutions of the outer surface on the inferior surface of the frontal lobe. Near the central line is the olfactory groove, which lodges the olfactory bulb and its peduncle.

The Olfactory Lobe.—This lobe consists of the olfactory bulb and its peduncle, the olfactory area of Broca, the trigonum olfactorium, and the anterior perforated space. The olfactory bulb is a club-shaped body, which gives off numerous nerve-fibers that pierce the dura and cribriform plate of the ethmoid bone. Posteriorly it is continued into its peduncle, which, traced backward, divides into two white bands, the outer passing toward the fissure of Sylvius, and the inner running to the side of the longitudinal fissure. Between these two limbs is a small elevation called the trigonum olfactorium, or the middle root of the olfactory peduncle. The area lying anterior and internal to the inner limb is known as the olfactory area of Broca. The anterior perforated space is the area situated between the external and the internal limb of the olfactory peduncle (Fig. 45).

The **parietal lobe** is situated immediately posterior to the frontal lobe, from which it is separated by the fissure of Rolando. Internally it is limited by the great longitudinal fissure, and posteriorly the parieto-occipital fissure separates it from the occipital lobe. Inferiorly the fissure of Sylvius separates it from the temporal lobe. Its posterior lateral surface is not separated from the occipital lobe by a definite fissure; its lateral surface is marked by two distinct fissures—the postcentral fissure, which is parallel to the fissure of Rolando, and the intraparietal fissure, which arches from before backward, and frequently communicates with the postcentral fissure.

These fissures divide the lateral surface of the parietal lobe into three well-marked divisions—the postcentral convolution, situated posterior to the fissure of Rolando; the superior and inferior parietal lobules, situated above and below the intraparietal fissure. The inferior parietal lobule is further divided into three parts—an anterior part, arched around the posterior end of the fissure of Sylvius, is called the supramarginal gyrus; a middle part, known as the angular gyrus, which arches around the posterior end of the superior temporal fissure, and a posterior part, called the postparietal gyrus, which is posterior to the angular gyrus.

The Occipital Lobe.—This lobe is pyramidal in shape, with its apex directed backward. It occupies the posterior portion of the hemisphere, and has three surfaces: an external, an internal, and an inferior. The external and internal surfaces are separated from the parietal lobe by the parieto-occipital fissure and by a line projected downward from this fissure across the external surface of the hemisphere. The lower anterior and inferior surfaces of his lobe are not distinctly separated from the parietal and temporal lobes. The outer surface is divided by two longitudinal fissures into a superior, a middle, and an inferior occipital convolution.

The **temporal lobe** occupies the middle fossa of the skull. It is conical in shape, and presents three surfaces: an upper, a lower, and an external or lateral. The temporal lobe is separated from the parietal lobe by the fissure of Sylvius. The external surface is limited above by this fissure and below by the inferior temporal sulcus. Posteriorly, this surface fuses with the occipital lobe. It is marked by three anteroposterior fissures—the superior, the middle, and the inferior—which separate this surface into a superior, middle, and inferior temporal convolution. The upper surface is hidden within the fissure of Sylvius. The inferior surface presents posteriorly a transverse depression, which serves as a dividing line between the temporal and occipital lobes. The inferior surfaces of the temporal and occipital lobes are separated into a superior and an inferior occipitotemporal convolution; these are separated from each other by the collateral fissure.

The **central lobe,** or island of Reil, lies deeply within the fissure of Sylvius. It is pyramidal is shape, and consists of five or six convolutions.

The Fissures and Convolutions of the Median Surface of the Hemispheres.—*Fissure of the Corpus Callosum.*—This fissure commences in front, below the anterior end of the corpus callosum. It runs along the upper margin of this structure, turns around its posterior extremity along the inner border of the temporal lobe, and extends to the notch of the uncus (Fig. 43).

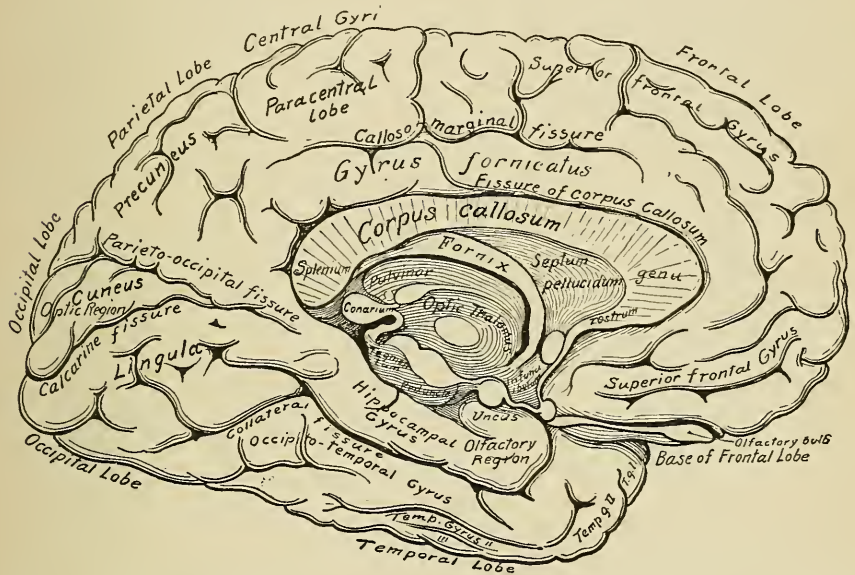


Fig. 43.—Fissures and convolutions of the median surface of the hemispheres.

The *callosomarginal fissure* commences in front, beneath the anterior end of the corpus callosum. It first extends forward, and then runs upward and backward, parallel to the margin of the hemisphere. It lies between the fissure of the corpus callosum and the upper margin of the hemisphere, and extends backward to a point corresponding to about the posterior end of the corpus callosum, where it turns upward to the upper border of the hemisphere, a little behind the fissure of Rolando. This fissure limits the extent of the frontal lobe on the median surface of the hemisphere.

The *fissura subparietalis* is the continuation of the callosomar-

ginal fissure from the point where it turns upward. This fissure marks the limit of the parietal lobe on the median aspect of the hemisphere.

The *parieto-occipital fissure* is well marked on the median aspect of the hemisphere. It is directed downward and forward, and merges with the calcarine fissure. It separates the median surface of the parietal lobe from the median aspect of the occipital lobe, and is continued for a short distance over on the lateral aspect of the cerebral hemisphere.

The *calcarine fissure* is a well-marked depression upon the median aspect of the occipital lobe. It runs forward and slightly upward from the posterior border of the hemisphere to join the parieto-occipital fissure.

The *hippocampal* or *dentate fissure* is that portion of the fissure of the corpus callosum which occupies the internal surface of the temporal lobe.

Convolutions of the Median Aspect of the Hemispheres.—Situated in front of and above the callosomarginal fissure is the marginal convolution, which forms the median aspect of the frontal lobe. The posterior part of this convolution in front of the ascending limb of the callosomarginal fissure is called the paracentral convolution. Situated between the ascending limb of the callosomarginal fissure and the parieto-occipital fissure, above the subparietal fissure, is the præcuneus, or quadrate lobule, which represents the median aspect of the parietal lobe. Between the parieto-occipital fissure and the calcarine fissure lies the cuneus, or wedge-shaped convolution, which represents a portion of the median aspect of the occipital lobe.

The *gyrus fornicatus*, gyrus cinguli, or convolution of the corpus callosum, commences in front, below the anterior end of the corpus callosum. It arches upward and backward, then around the posterior end of the corpus callosum, where it becomes slightly constricted and is here called the isthmus. It then runs downward and forward on the internal aspect of the temporal lobe as the gyrus hippocampi, or superior occipitotemporal convolution, ending in front in the uncus of the temporal lobe. This group of convolutions, together with the structures included within their zone, forms the **limbic lobe**.

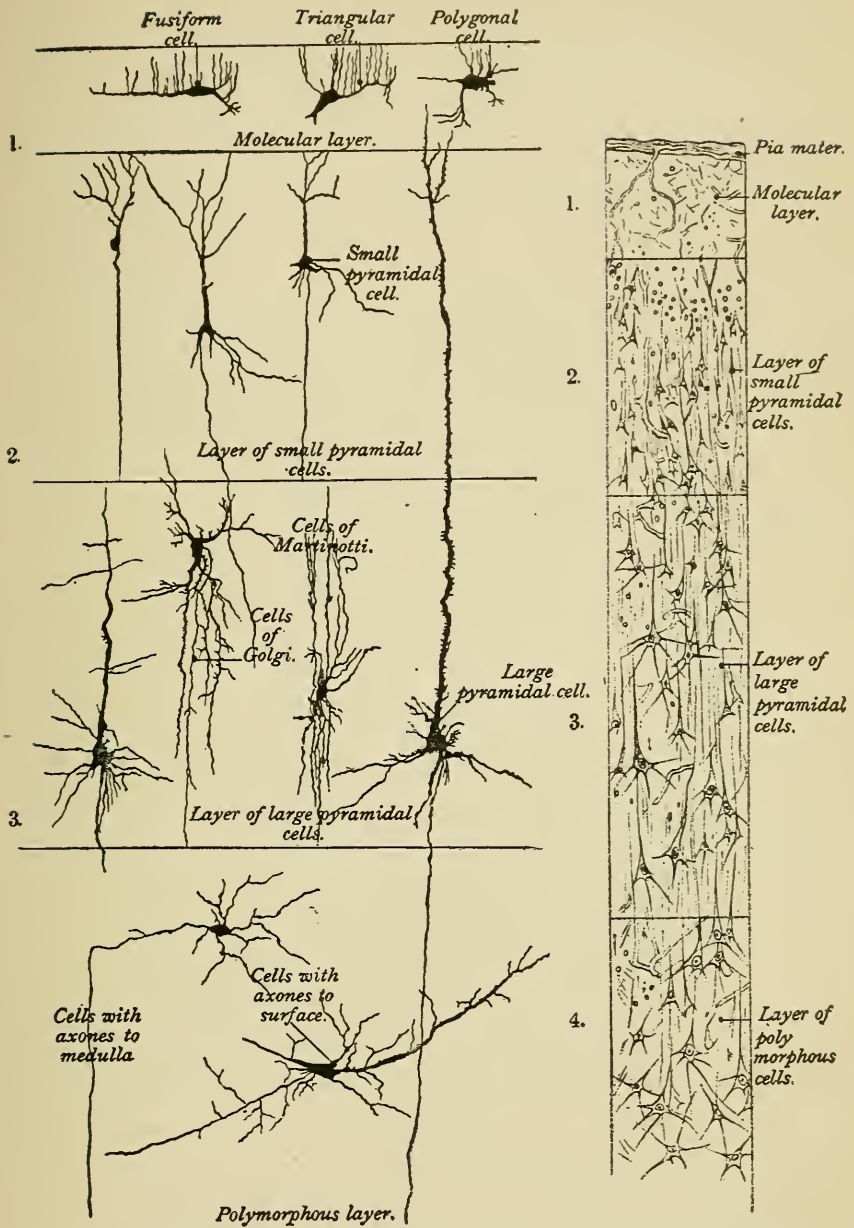


Fig. 44.—Microscopic structure of the cortex of the cerebrum. (Whitaker, "Anatomy of the Brain and Spinal Cord," after Ramon y Cajāl.)

The Structure of the Cortex of the Cerebrum (Fig. 44).—The gray matter of the cerebral cortex is arranged in several more

or less distinct layers. These are composed of nerve-cells and nerve-fibers, neuroglia, blood-vessels, and lymphatics. There are four more or less distinct layers of the cortex which are characterized by definite types of cells: (1) The molecular layer, which contains many neuroglia cells, polygonal, fusiform, and triangular cells. The axis-cylinder processes of these cells are almost entirely confined to this layer. (2) A layer of small pyramidal cells. (3) A layer of large pyramidal cells, called the cells of Betz. These cells are large, thin axis-cylinder processes, descending, and in the upper part of their course giving off many branches. The process then descends, and ultimately ends in the gray matter at the lower levels. (4) A layer of polymorphous cells. In this layer the cells vary much in size and shape. In the second, third, and fourth layers are also found the cells of Golgi and the cells of Martinotti.

The character and type of cells vary in different regions of the cerebral cortex. In some regions eight distinct layers may be recognized, whereas in others it is difficult accurately to identify four layers.

The white and gray fibers found in the cortex are the processes of the nerve-cells of the cortex and of those that have ascended from lower levels.

Neuroglia pervades the entire cortex, and is specially well marked along the blood-vessels and immediately beneath the pia.

THE UNDER ASPECT OF THE CEREBRUM

The under aspect of the brain is irregular in shape. It presents for examination the cerebellum; the medulla; the pons Varolii; the cerebral peduncles; the inferior aspect of the frontal and temporal lobes; the anterior part of the great longitudinal fissure; the internal portion of the fissure of Sylvius; the circle of Willis; the interpeduncular space; and the superficial origins of the cranial nerves.

A careful study of the accompanying illustration (Fig. 45) will reveal the relation of these various structures to one another. Each of the structures has been or will be described under its appropriate head.

The **interpeduncular space** is an irregular area at the base of the brain, situated between the great longitudinal fissure anteriorly, the under surface of the frontal and temporal lobe on each side, and the cerebral peduncle posteriorly. This irregular-shaped interval is formed by a thin layer of gray and white matter stretching across the middle line, uniting the under surface of the two hemispheres.

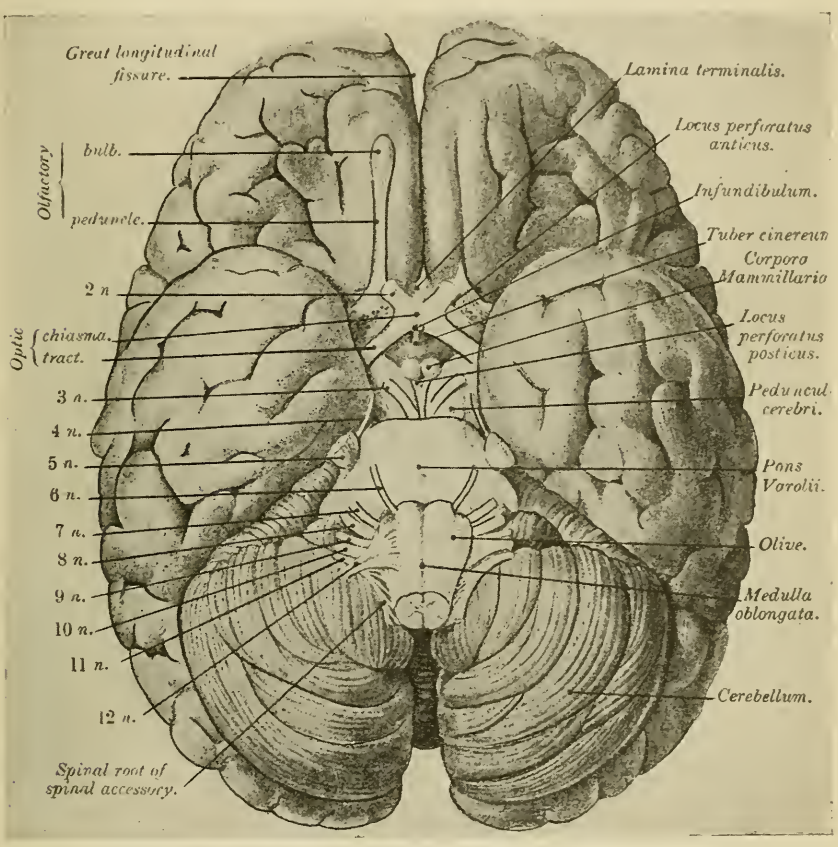


Fig. 45.—Base of the brain. (Whitaker, "Anatomy of the Brain and Spinal Cord.")

The **anterior perforated space** is a grayish lamina, perforated for the passage of blood-vessels into the brain. It is situated external to the optic commissure, at the root of the fissure of Sylvius, behind and between the roots of the olfactory peduncles. Externally it is continuous with the hemispheres.

The **posterior perforated space,** called also the pons Tarini, is a gray lamina, similar to the anterior perforated space, and, like it, perforated for the passage of blood-vessels into the interior of the cerebrum. It is situated between the diverging cerebral peduncles, and posterior to the tuber cinereum.

The Pituitary Body, or Hypophysis Cerebri.—This gland is oval in shape, and has a reddish color. It consists of two portions—the anterior lobe, which is made up of a network of connective tissue and of epithelial cells, and is developed originally from the buccal cavity; and a posterior lobe, which consists of a network of fine nerve-fibers, connective tissue, and neuroglia. The posterior lobe is developed originally from the anterior cerebral vesicle, and is connected with the third ventricle by the infundibulum. Between the anterior and posterior lobes, and connecting them, is the pars intermedia. In the pars intermedia are found cells that secrete a colloid substance, resembling in appearance the characteristic secretion of the thyroid gland. The posterior lobe is poorly supplied with blood-vessels, whereas the anterior lobe is highly vascular.

The **corpora albicantia** are rounded, pea-shaped nodules, situated in front of the posterior perforated space, between it and the tuber cinereum. These small white bodies are connected with the fornix.

The Tuber Cinereum.—This is a somewhat irregular-shaped elevation, situated anterior to the corpora albicantia. Projecting downward from the tuber cinereum is a funnel-shaped process called the infundibulum. Attached to its inferior end is an egg-shaped gray mass, the *hypophysis cerebri*, which is situated in the sella turcica. The posterior lobe of this gland is alone connected with the infundibulum.

The Optic Chiasm or Commissure and the Optic Tracts.—The optic commissure is quadrilateral in form, and rests upon the optic groove of the sphenoid bone. On either side of the optic commissure is the anterior perforated space, and posterior to it is the tuber cinereum. Within the commissure the optic nerves of the two sides undergo partial decussation. Anteriorly, the optic commissure divides into the two optic nerves, which become rounded, and are inclosed in a sheath derived from the

arachnoid, which is covered by a sheath from the dura. The optic nerves pursue an anterior course, and enter the orbit through the optic foramina.

The **optic tracts** are two well-marked, flattened bundles of fibers that are given off from the posterior portion of the optic commissure. They run backward and outward, passing obliquely across the surface of the crusta in the form of a flattened band. They then divide into two parts—an external and an internal. The external part enters the external geniculate body and the under surface of the pulvinar of the optic thalamus; it receives most of the fibers of the brachium of the superior corpora quadrigemina. The internal division enters the internal geniculate body, from which it derives its fibers.

As has been stated, the optic nerves of the two sides undergo a partial decussation within the optic commissure. The fibers that form the inner margin of each tract are continued across from one side of the brain to the other (commissure of Gudden). The outer fibers of each tract are continued into the optic nerve of the same side. The central fibers of each tract are continued through the commissure to the optic nerve of the opposite side.

INTERIOR OF THE CEREBRUM

Upon section of the brain the following structures are encountered: Immediately beneath the cortex the hemispheres consist of a mass of white tissue that is made up of medullated fibers of varying size. They may be divided into three distinct systems, according to the course which they pursue: projection fibers, commissural fibers, and association fibers. Cavities, which are called ventricles, and collections of gray masses, the basal ganglia, are also seen (Fig. 46).

The **ventricles** of the brain are five in number. They are the remains of the neural canal, from the walls of which the brain and the spinal cord are developed. Upon coronal section they are seen to be somewhat T shaped. The ventricles, with the exception of the fifth, communicate with one another and with the subarachnoid space.

The **lateral ventricles,** or first and second ventricles, are two irregular, crescent-shaped cavities, hollowed out in the sub-

stance of the cerebral hemisphere. Each of these ventricles consists of a central part, the body, and of three prolongations—the anterior, the posterior, and the descending or lateral horns. The roof is formed by the corpus callosum, and the floor by a small part of the corpus callosum, the nucleus caudatus, the optic thalamus, the stria terminalis, the choroid plexus, and the free edge of the body of the fornix. In front, the inner wall of

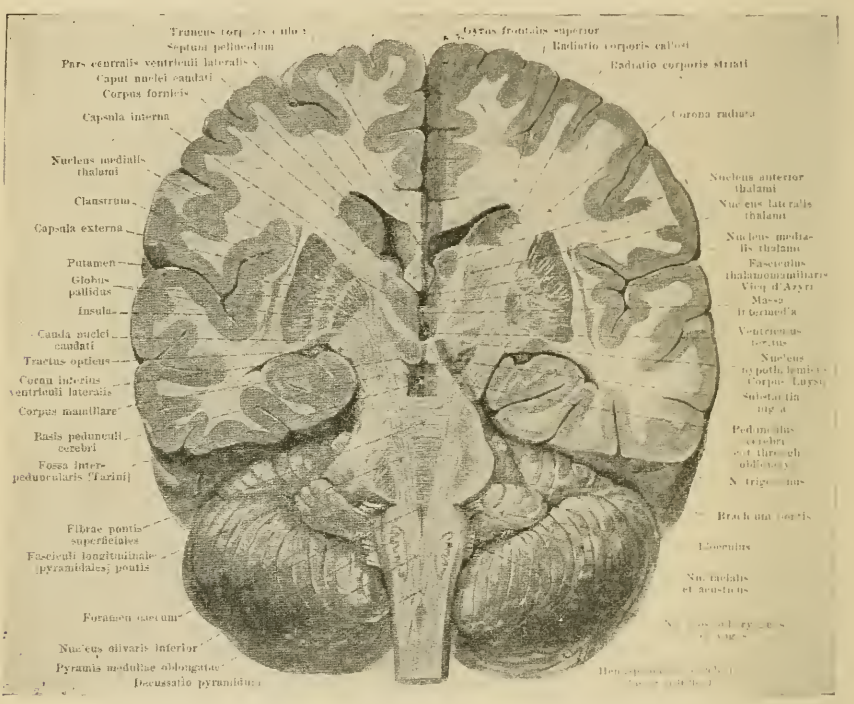


Fig. 46.—Section through the brain in direction of the cerebral peduncles. (After Spalteholz.)

the ventricle is formed by a thin, double, vertical triangular partition, the *septum lucidum*, which extends between the under surface of the corpus callosum and the upper surface of the fornix. Externally the floor and roof meet, so that there is no outer wall (Fig. 47).

The *anterior horn* is short, and turns forward and outward from the anterior part of the body of the ventricle into the substance of the frontal lobe.

The *posterior horn* passes backward, outward, and inward into the substance of the occipital lobe. On the floor of this horn is an oval prominence called the *hippocampus minor*, caused by the calcarine fissure. Above this is a slight ridge, produced

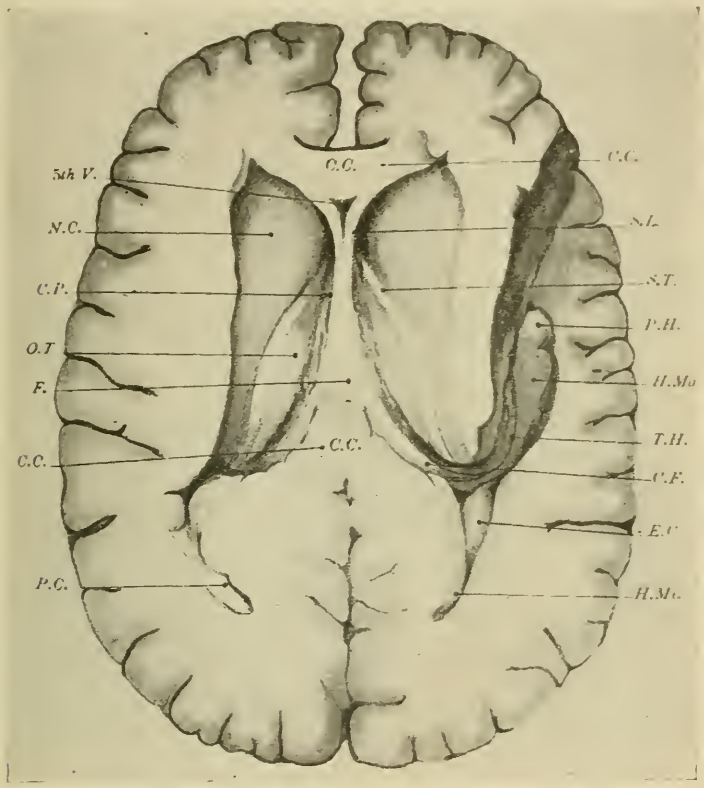


Fig. 47.—Lateral ventricle: 5th *V.*, Fifth ventricle; *N.C.*, nucleus caudatus; *C.P.*, choroid plexus; *O.T.*, optic thalamus; *F.*, fornix; *C.C.*, corpus callosum; *P.C.*, posterior cornu; *S.T.*, stria terminalis; *S.L.*, septum lucidum; *P.H.*, pes hippocampi; *H.Ma.*, hippocampus major; *T.H.*, tinea hippocampi; *C.F.*, crura fornicis; *E.C.*, eminentia collateralis; *H.Mi.*, hippocampus minor. (Whitaker, "Anatomy of the Brain and Spinal Cord.")

by radiating bundles of the corpus callosum, and known as the *forceps minor*.

The *lateral*, *descending*, or *inferior horn* curves around the posterior end of the optic thalamus, runs backward, outward, and then downward and forward, and finally turns inward in the substance of the temporal lobe. In the floor of this horn is

an elongated projection following the bend of the horn, and called the *hippocampus major*. This elevation is caused by the hippocampal fissure. The choroid plexus of the lateral ventricle projects into this horn.

The lateral ventricles are lined by a layer of gray matter, covered on the surface by columnar ciliated epithelium, which is continuous with the epithelium of the other ventricles.

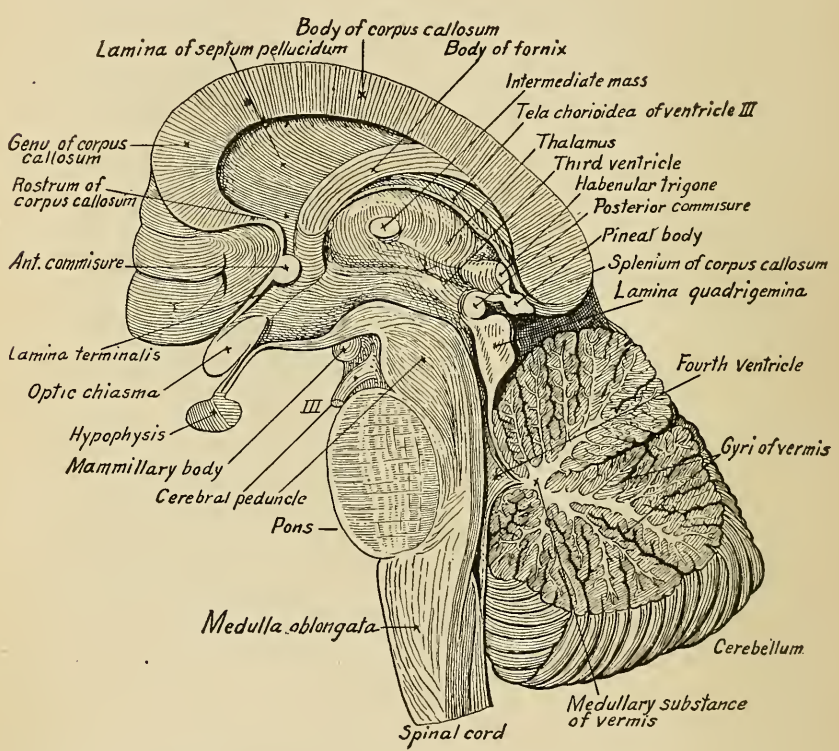


Fig. 48.—Median section of the brain without the hemispheres.

The Foramen of Monro.—Between the fornix in front and the optic thalami behind is a slit-like opening by means of which the lateral ventricles communicate with the third ventricle, and indirectly with each other; this is called the foramen of Monro. Through this foramen the choroid plexus of the lateral ventricle is continuous with that of the third ventricle.

The **third ventricle** is a narrow, vertical cleft, situated in the

middle line of the cerebrum, between the two optic thalami. It lies beneath the body of the fornix, and extends down to the base of the brain, being deeper in front than behind. Its roof is formed by the under surface of the velum interpositum, which

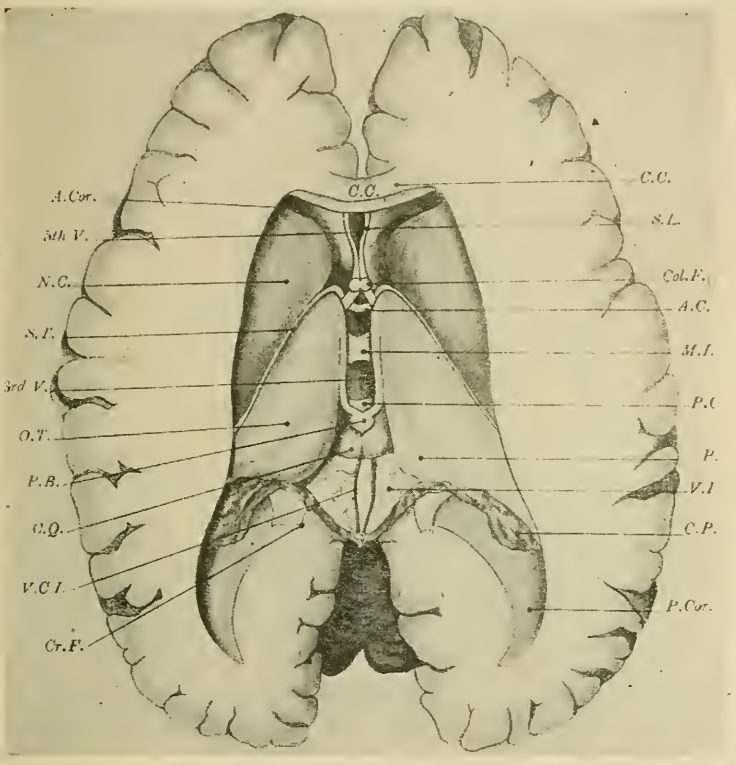


Fig. 49.—Third ventricle, from above: *A.Cor.*, anterior cornu; *5th V.*, fifth ventricle; *N.C.*, nucleus caudatus; *S.T.*, stria terminalis; *3rd V.*, third ventricle; *O.T.*, optic thalamus; *P.B.*, pineal body; *C.Q.*, corpora quadrigemina; *V.C.I.*, venæ cerebri internæ; *Cr.F.*, crura fornicis; *C.C.*, corpus callosum; *S.L.*, septum lucidum; *Col.F.*, columnæ fornicis; *A.C.*, anterior commissure; *M.I.*, massa intermedia; *P.C.*, posterior commissure; *P.*, pulvinar; *V.I.*, velum interpositum; *C.P.*, choroid plexus; *P.Cor.*, posterior cornu. (Whitaker, "Anatomy of the Brain and Spinal Cord.")

is covered by a layer of flattened epithelial cells. Its floor is formed by the dorsal part of the cerebral peduncles, the posterior perforated space, the corpora albicantia, the tuber cinereum, the infundibulum, and the optic chiasm. In front, the ventricle is limited by the anterior pillars of the fornix, and behind it

opens into the aqueduct of Sylvius. The lateral walls are formed by the inner surfaces of the optic thalami. Across the center of the third ventricle, between the optic thalami, is a small gray band called the middle or gray commissure. The third ventricle is lined by epithelium similar to and continuous with that of the other ventricles. Hanging from the roof along the middle line, from the under surface of the velum interpositum, are the folds of the choroid plexuses. The third ventricle communicates in front and above, through the foramen of Monro, with the lateral ventricles; posteriorly, through the aqueduct of Sylvius, with the fourth ventricle; in the anterior part, through a conical-shaped passage in the floor, with the infundibulum (Figs. 48 and 49).

The **septum lucidum** is a thin, double, vertical partition, which separates the lateral ventricles from each other. It is triangular in shape, and fills up the interval between the inner, knee-shaped bend of the corpus callosum and the anterior pillars of the fornix. Its apex is directed posteriorly. Between its two layers is a narrow, slit-like cavity, called the *fifth ventricle.* On the internal surface of each lamina of the septum is a thin layer of gray matter, and on the exterior surface, next to the cavity of the lateral ventricles, is a layer of white matter covered by the neuroglia and epithelium. The cavity of the fifth ventricle is not lined by epithelium, and does not communicate with any of the other ventricles (Fig. 48).

The **velum interpositum** is a fold of pia mater that enters the brain through the great transverse fissure of the cerebrum, appearing in the third and lateral ventricles. It is covered by ependymal epithelium. It is triangular in shape, with its apex directed forward, and reaches from the foramen of Monro in front to the back part of the splenium of the corpus callosum, beneath which, after investing the pineal gland, it is continuous with the pia mater of the cerebrum and cerebellum.

At the sides, the free edges of the velum interpositum project into the floor of the lateral ventricles, and rest on the upper surfaces of the optic thalami, passing into the descending horns of the lateral ventricles. In each lateral margin of the velum interpositum are vascular fringes that consist of tortuous ramifications of small blood-vessels, forming the choroid plexuses of

the lateral ventricles which extend to the apices of the descending horns. As these vascular fringes pass backward they send a projection into the posterior horns. They are continuous through the foramen of Monro into the third ventricle, projecting downward, along the middle line of the under aspect of the velum interpositum, to form the choroid plexuses of the third ventricle (Fig. 50).

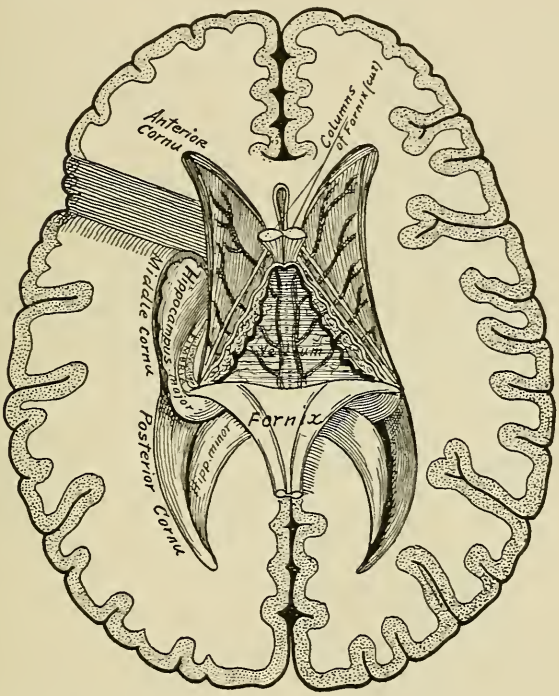


Fig. 50.—Horizontal section of cerebral hemispheres; fornix has been reflected to show the velum interpositum.

The **internal cerebral veins** are two veins that gather up the blood from the choroid plexuses and the basal ganglia, and run backward side by side between the layers of the velum interpositum. They unite to form the vein of Galeni, which enters the straight sinus.

The great transverse fissure of the brain is horseshoe shaped, its central portion corresponding to the base of the fornix, and its lateral part to the descending horns of the lateral ventricles.

Through this fissure the pia mater and choroid plexuses project into the ventricles.

The Basal Ganglia.—These are large gray masses situated at the base of the brain. The *corpora striata* are two in number, one in each hemisphere. Each corpus is divided into two parts—an intraventricular portion and an extraventricular portion. The

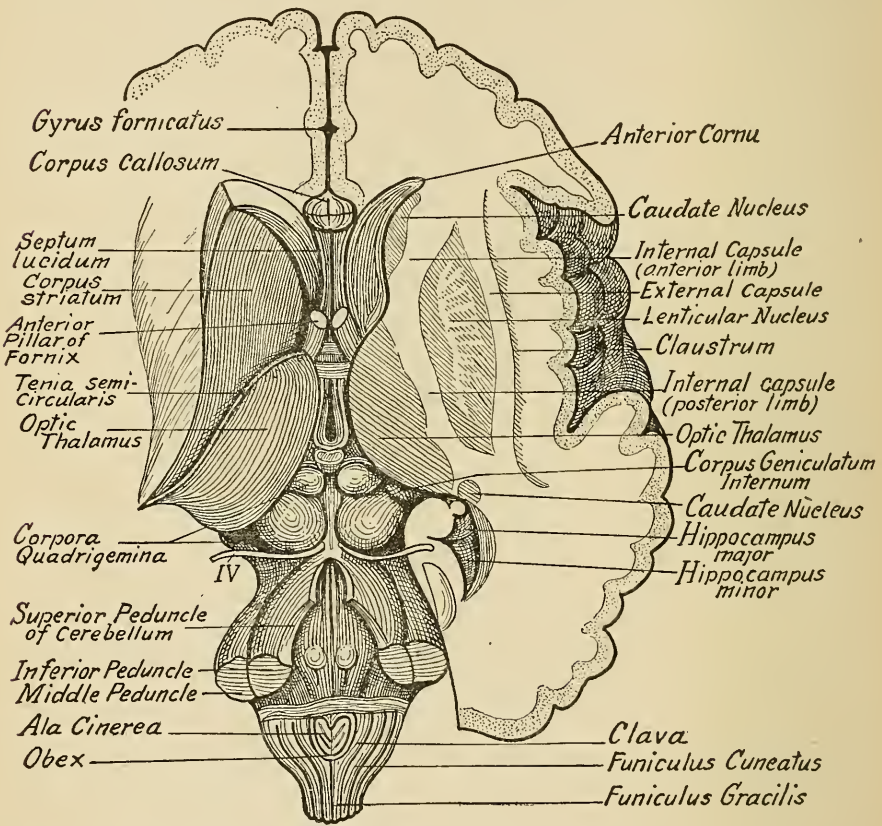


Fig. 51.—Horizontal section of cerebrum, showing basal ganglia, optic thalamus, etc,

nucleus caudatus, the intraventricular portion of the corpus striatum, is pear shaped, and has a pinkish-gray color. It consists of a gray core streaked with white fibers, and covered on the surface by layers of white substance. Its larger end or head is directed forward, gradually tapering as it passes backward to the outer side of the optic thalamus, where it turns down-

ward, outward, and forward into the roof of the descending horn of the lateral ventricle. It can be traced as far forward as the tip of the temporal lobe. In its course it forms an arch with its concavity directed forward.

The *lenticular nucleus*, the extraventricular part of the corpus striatum, is placed externally to both the caudate nucleus and the optic thalamus, being separated from them by a strand of white nerve-fibers called the internal capsule. On horizontal section it is seen to be shaped like a double convex lens. It is divided vertically by two white lamina into three parts—an internal, a middle, and an external. These differ somewhat in color, the internal and middle portions, being of a paler hue, are called the *globus pallidus*. The external portion is called the *putamen*. In front and below the caudate nucleus and the lenticular nucleus are continuous with each other and with the gray matter of the anterior perforated space (Fig. 51).

The *optic thalami* are two large, oval prominences, situated above the cerebral peduncles, behind and internal to the corpora striata. Each optic thalamus consists of a central gray core covered by a stratum of white matter. Each thalamus has four surfaces and two ends. The anterior end is rounded, and the posterior end swells out into a prominence called the *pulvinar*. The gray matter of the optic thalami is arranged as three nuclei—anterior, outer, and inner (Figs. 51 and 52).

Functions.—These ganglia, together with the corpora striata, are very probably intermediate stations for the transmission of sensation and motion. They also exert some influence on the reflex activities of the body, and it is highly probable that it is through these structures that the complicated and instantaneous reflexes that occur as a consequence of visual and auditory stimulation take place.

The Geniculate Bodies.—Below and external to the pulvinar of each optic thalamus are two oval prominences—the internal and external geniculate bodies. The internal body is smaller, and is separated from the external by a band of white fibers which is one of the roots of the optic tract.

The geniculate bodies consist of a gray core and a white cortex. The external geniculate body is connected by a band of fibers to

the anterior, and the internal geniculate body to the posterior, corpora quadrigemina. The external geniculate body is also connected with the optic tracts.

The **pineal gland** is a reddish, vascular, oval body, situated in the anterior part of the longitudinal groove, between the superior corpora quadrigemina. It is composed of follicles separated by connective tissue, and filled with epithelial cells and calcareous particles (Figs. 52 and 53).

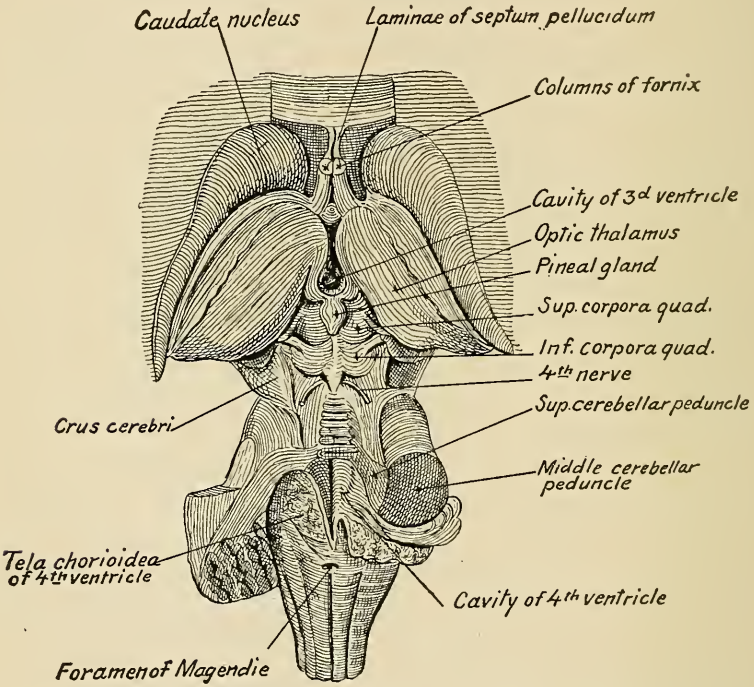


Fig. 52.—The thalami, the epithalamus, the lamina quadrigemina, and the rhombencephalon, seen from behind and above, after removal of the greater part of the cerebellum.

The **corpus callosum** is a longitudinal band of transverse fibers that arches from before backward, in the middle line, between the two cerebral hemispheres, which it connects. It is about four inches long, and forms the floor of the great longitudinal fissure. It approaches the anterior aspect of the brain more nearly than the posterior. It is thicker at the ends than in the middle, and is thickest and widest behind. Anteriorly

it turns downward and backward upon itself, making a knee-shaped bend called the *genu*. Gradually becoming smaller, it forms a narrow median band, the rostrum, which, at the base of the brain, bifurcates into two tapering processes, known as the peduncles of the corpus callosum. These can be traced into the anterior perforated spaces at the root of the fissure of Sylvius. Posteriorly, it ends in a thickened, free, rounded border—the splenium. At the outer ends of the splenium two horn-shaped

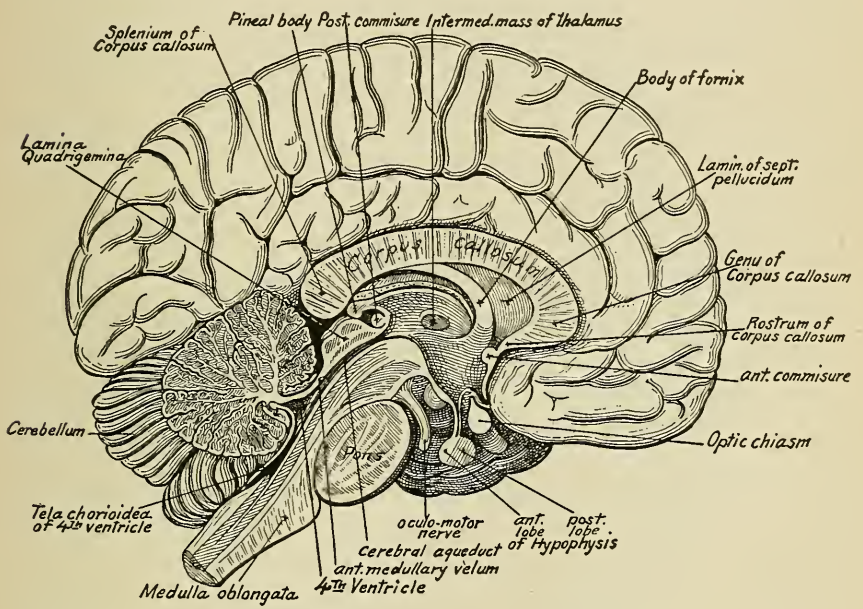


Fig. 53.—Median section of the brain.

bundles of fibers—the forceps major—diverge into the occipital lobes (Fig. 53).

The upper surface of the corpus callosum is covered by a thin layer of gray matter. It is marked in the middle line by a longitudinal groove, *the raphe*, parallel to which, on each side, are seen faint lines—the median longitudinal striæ or nerves of Lancisi. External to these, under the overhanging edge of the gyrus fornicatus, are the lateral longitudinal striæ. The under surface of the corpus callosum in the middle line, in the posterior half of its extent, rests upon the body of the fornix, to which it is

closely adherent. Laterally the transverse fibers of the corpus callosum form the roof of the lateral ventricles, and then spread out into the white substance of the hemispheres (Fig. 54).

The corpus callosum consists principally of transverse fibers, only a few being longitudinal. They pass into the hemispheres, diverge in all directions, go to the gray matter of the cortex, and connect similarly situated areas with each other.

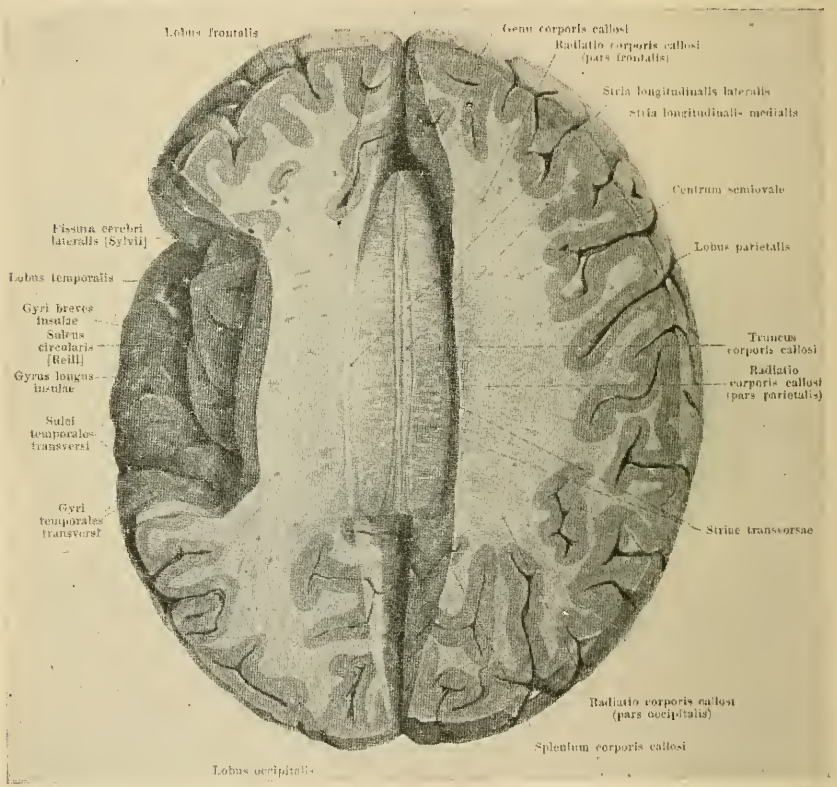


Fig. 54.—Corpus callosum viewed from above (Spalteholz).

The **anterior commissure** is a small, round, twisted bundle of transverse white fibers situated in front of the anterior pillars of the fornix. It connects the two olfactory lobes and the two temporal lobes. (See Fig. 48.)

The **middle** or **gray commissure** is a delicate band of gray matter crossing the third ventricle, and connected with the gray matter of the optic thalami. (See Fig. 48.)

The **posterior commissure** is a small band of white fibers situated in front of and below the pineal gland, above the aqueduct of Sylvius. (See Fig. 48.)

The corpus callosum and the anterior, middle, and posterior commissures are the pathways which connect similarly situated areas of the hemispheres.

The **fornix** is an arched white band that can be traced from the tip of the descending horn of the lateral ventricle to the corpora albicantia. The fibers that go to make up the fornix arise as two ribbon-like bands, one on each side, from the free surface of the hippocampus major. They ascend from the free surface of the hippocampus major, wind around the posterior ends of the optic thalami, where they become the crura fornicis. They next converge toward each other and meet in the middle line, to form a white, flattened, triangular-shaped body,—the body of the fornix,—which has its apex directed forward, and is blended above and posteriorly with the corpus callosum. Inferiorly, it lies upon the velum interpositum, which separates it from the optic thalami and the third ventricle. Its fibers then diverge and go downward and forward as two round, finger-like processes, called the anterior pillars of the fornix. These lie side by side, and can be traced forward to the base of the brain to the corpora albicantia. In this position they make a figure-of-8-shaped twist upon themselves, forming the cortex of these structures, and end in gray matter. From cells in the gray matter of the corpora albicantia new fibers arise; these are termed the *bundle of Vicq d'Azyr*. The anterior pillars of the fornix, as they descend in front of the optic thalami, help to form the anterior boundary of the foramen of Monro. The body of the fornix enters into the formation of the floor of the lateral ventricles. (See Figs. 47 and 48.)

The *fornix* is a tract of association-fibers. These fibers connect different parts of the same hemisphere.

There are numerous other association-fibers which unite adjacent and distant convolutions with each other (Fig. 55).

The principal **projection pathways** are contained in the internal and external capsule. They are made up of fibers that connect

the gray matter of the hemispheres with the lower part of the cerebrospinal axis (Fig. 56).

Upon horizontal section the **internal capsule** appears as a longitudinal, semilunar-shaped band of white matter, with its concavity directed outward. It may be divided into three portions—an anterior, a middle, and a posterior. The anterior division lies between the caudate nucleus internally and the lenticular nucleus externally. The posterior division lies be-

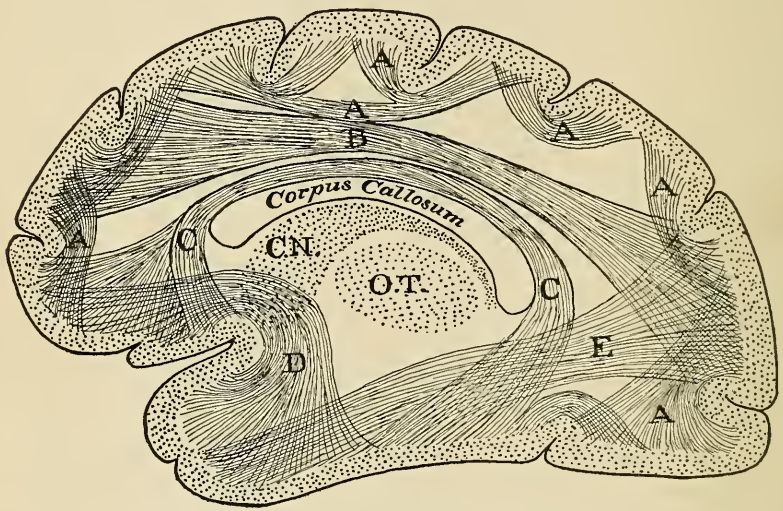


Fig. 55.—Lateral view of a human hemisphere, showing the bundles of association-fibers: *A*, *A*, Between adjacent gyri; *B*, between frontal and occipital areas; C, between frontal and temporal areas, cingulum; *D*, between frontal and temporal areas, fasciculus uncinatus; *E*, between occipital and temporal areas, fasciculus longitudinalis inferior; *C.N.*, caudate nucleus; *O.T.*, thalamus (Starr).

tween the optic thalamus internally and the lenticular nucleus externally. The anterior and posterior divisions form a sharp angle or knee. As they ascend, the fibers of the internal capsule form a radiating, fan-shaped group that aid in the formation of the corona radiata, which spreads out to the cerebral cortex (Figs. 56 and 57).

Functions of the Internal Capsule.—The anterior limb is made up of fibers which ascend and descend from the anterior part of the frontal lobe, to terminate or originate in the gray matter of

the basal ganglia and the pons. The knee and the anterior two-thirds of the posterior limb are made up of fibers which arise in the posterior part of the frontal lobe, and terminate in the parts below; they transmit impulses that result in voluntary

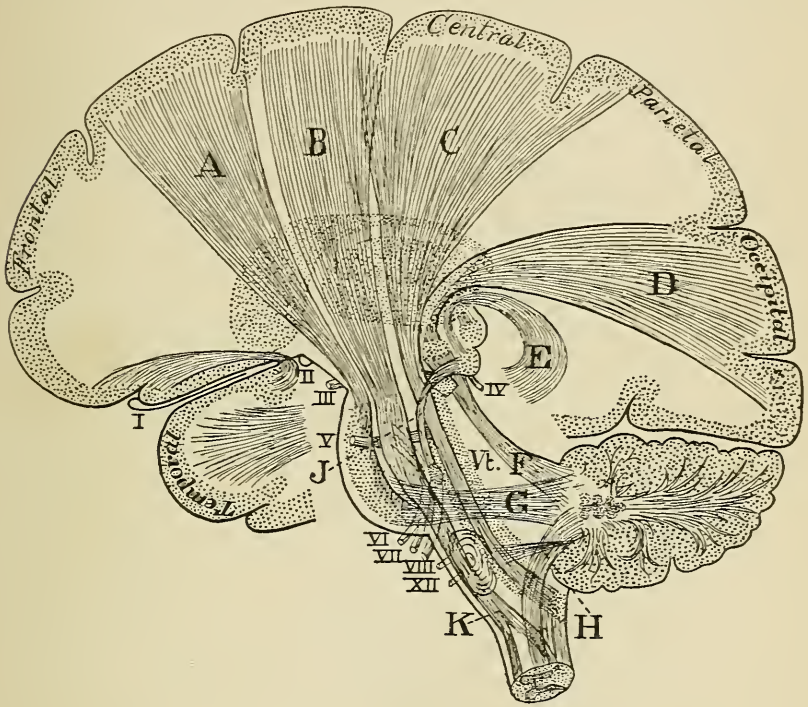


Fig. 56.—Schema of the projection fibers of the cerebrum and of the peduncles of the cerebellum; lateral view of the internal capsule: *A*, Tract from the frontal gyri to the pons nuclei, and so to the cerebellum (frontal cerebrocorticopontal tract); *B*, the motor (pyramidal) tract; *C*, the sensory (lemniscus) tract; *D*, the visual tract; *E*, the auditory tract; *F*, the fibers of the superior peduncle of the cerebellum; *G*, fibers of the middle peduncle uniting with *A* in the pons; *H*, fibers of the inferior peduncle of the cerebellum; *J*, fibers between the auditory nucleus and the inferior colliculus; *K*, motor (pyramidal) decussation in the bulb; *Vt.*, fourth ventricle. The numerals refer to the cranial nerves. (Modified from Starr.)

motion, and are placed in the following order: From before backward, those that have to do with the muscles of the eyes, then the mouth and tongue, face, upper extermities, legs, and trunk. These fibers descend and influence structures situated on the opposite side of the crura, pons, medulla, and spinal cord.

Blended with these fibers are secondary motor pathways that connect the central convolutions with the pontile nuclei and the cerebellum of the opposite side.

The posterior third of the posterior limb is occupied by fibers that transmit sensory impulses upward. Behind these fibers

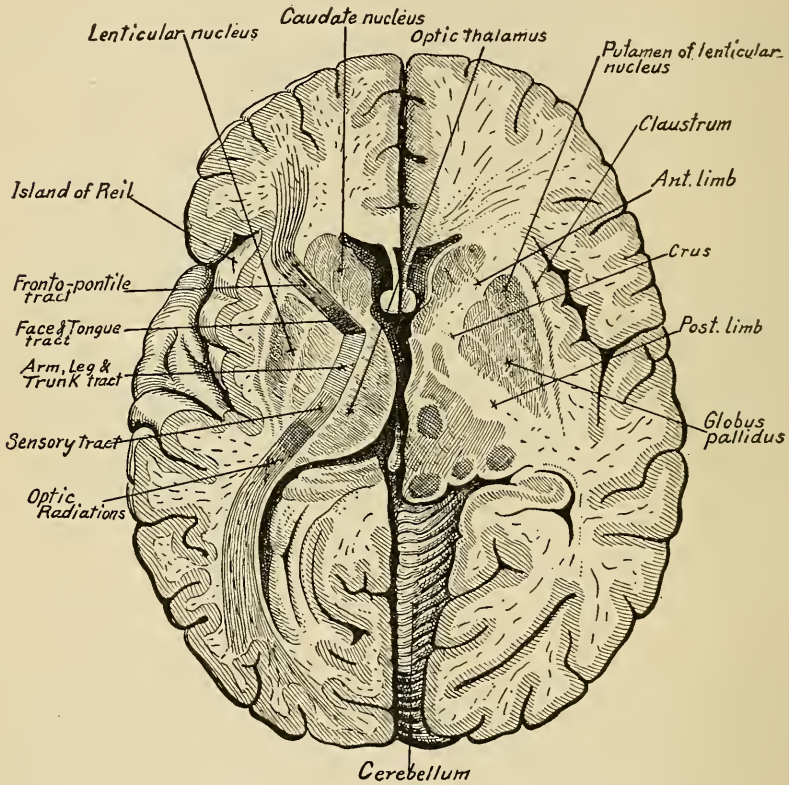


Fig. 57.—Horizontal section of the brain.

are fibers that connect the occipital and temporal lobes with the pontile nuclei, the optic thalamis, etc.

The *external capsule* is a thin, longitudinal strand of white matter which lies between the lenticular nucleus and the island of Reil. It consists of fibers whose course has not been determined, but they are believed to be association-fibers.

BLOOD-SUPPLY OF THE CRANIAL CONTENTS

Blood enters the cranial cavity principally by means of two vertebral and two internal carotid arteries. The two vertebral arteries enter through the foramen magnum, and pass forward and upward in front of the medulla to unite and form the basilar artery at the lower part of the pons Varolii. The vertebral arteries give off the posterior inferior cerebellar arteries, which wind backward around the upper part of the medulla to the under surface of the cerebellum. These vessels anastomose with the anterior inferior cerebellar and the superior cerebellar arteries, which are branches of the basilar artery; branches from the posterior inferior cerebellar go to the choroid plexus of the fourth ventricle. From the basilar arise transverse branches that supply the pons Varolii and adjacent parts of the brain. One branch, the internal auditory, accompanies the auditory nerve into the internal auditory meatus. Another branch, the anterior inferior cerebellar artery, is distributed to the anterior border of the inferior surface of the cerebellum. The superior cerebellar artery arises near the termination of the basilar, winds around the crura cerebri, and is distributed to the upper surface of the cerebellum. The posterior cerebral arteries are two terminal branches of the basilar. They wind around the crura cerebri, and pass to the surface of the occipital lobe of the cerebrum. Near their origin they receive the posterior communicating arteries from the internal carotid. Branches of the posterior cerebral and of the posterior communicating arteries enter the posterior perforated space and supply the inner surface of the optic thalamus, etc. (Fig. 58).

The Internal Carotids.—The two internal carotid arteries enter the cranial cavity through the **S**-shaped canal in the petrous portion of the temporal bone. Perforating the dura on the inner side of the anterior clinoid process, the internal carotid then enters the inner extremity of the fissure of Sylvius, where it gives off its cerebral branches. This portion of the artery has the optic nerve on its inner side and the third nerve externally. It divides into the anterior cerebral, the middle cerebral, the posterior communicating, and the anterior choroid arteries (Fig. 58).

The **anterior cerebral artery** passes forward in the great longitudinal fissure between the two anterior lobes of the brain, being connected, soon after its origin, with the vessels of the opposite side by a short anastomosing trunk—the anterior communicating. The two anterior cerebral arteries, lying side

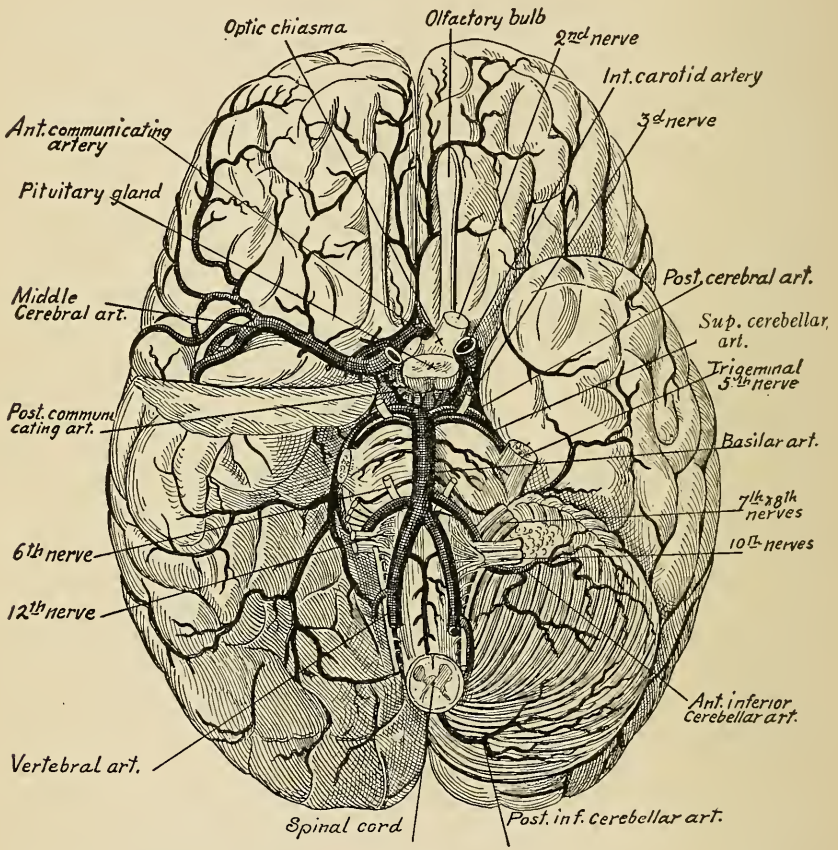


Fig. 58.—The arteries of the base of the brain.

by side, curve around the anterior part of the corpus callosum, and run along its upper surface to its posterior part, where they terminate by anastomosing with the posterior cerebral arteries. These vessels give off branches that pierce the anterior perforated space and supply the caudate nucleus. Branches are also given off which supply the two inferior frontal convolutions, the

corpus callosum, the inner surface of the first frontal and the upper part of the ascending frontal convolutions, and the medial aspect of the quadrate lobe (Fig. 59).

The **middle cerebral artery** is the largest branch of the internal carotid. It passes outward along the fissure of Sylvius over the island of Reil, and divides into terminal branches. It gives off many short branches which pierce the anterior perforated space and supply the caudate nucleus, the lenticular nucleus, the

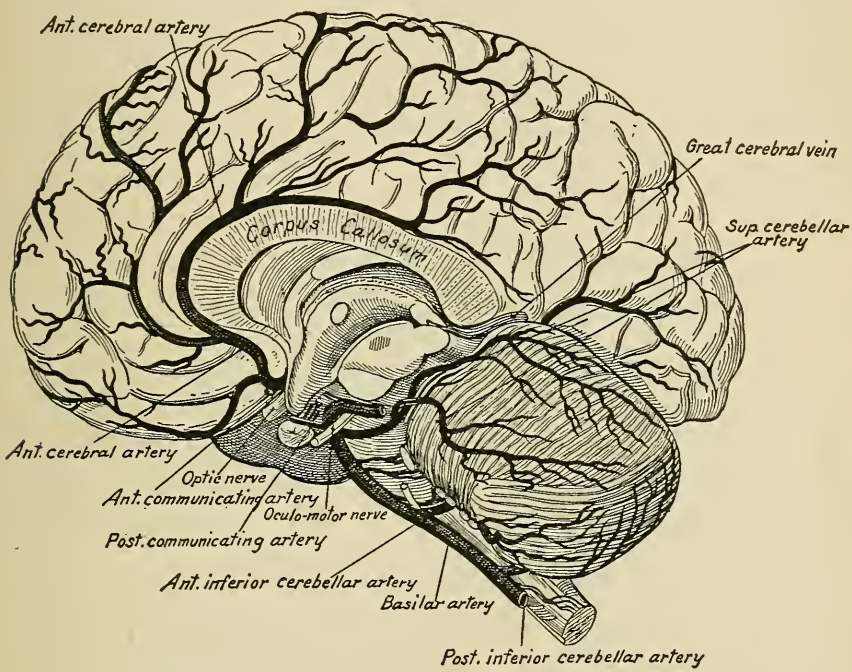


Fig. 59.—The arteries of the mesial surface of the cerebrum and of the surface of the cerebellum.

internal capsule, and part of the optic thalamus. One of these arteries, distributed to the lenticular nucleus, is of a larger size than the others. It is of special importance as being the artery in the brain that is most frequently ruptured. Charcot has termed it "the artery of cerebral hemorrhage" (Fig. 60). It also sends branches to the third ascending frontal, the parietal and the temporal lobes, and the angular gyrus (Fig. 61).

The *posterior communicating artery* arises from the back part

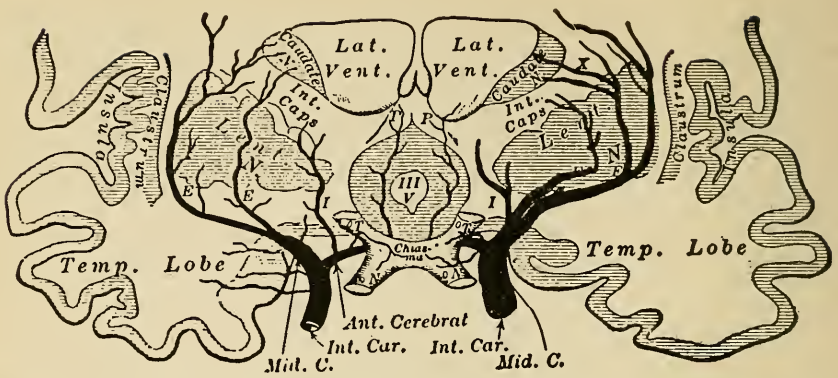


Fig. 60.—Showing distribution of blood-vessels to the internal capsule, artery marked *X* is the so-called "artery of hemorrhage." (Starr, after Duret.)

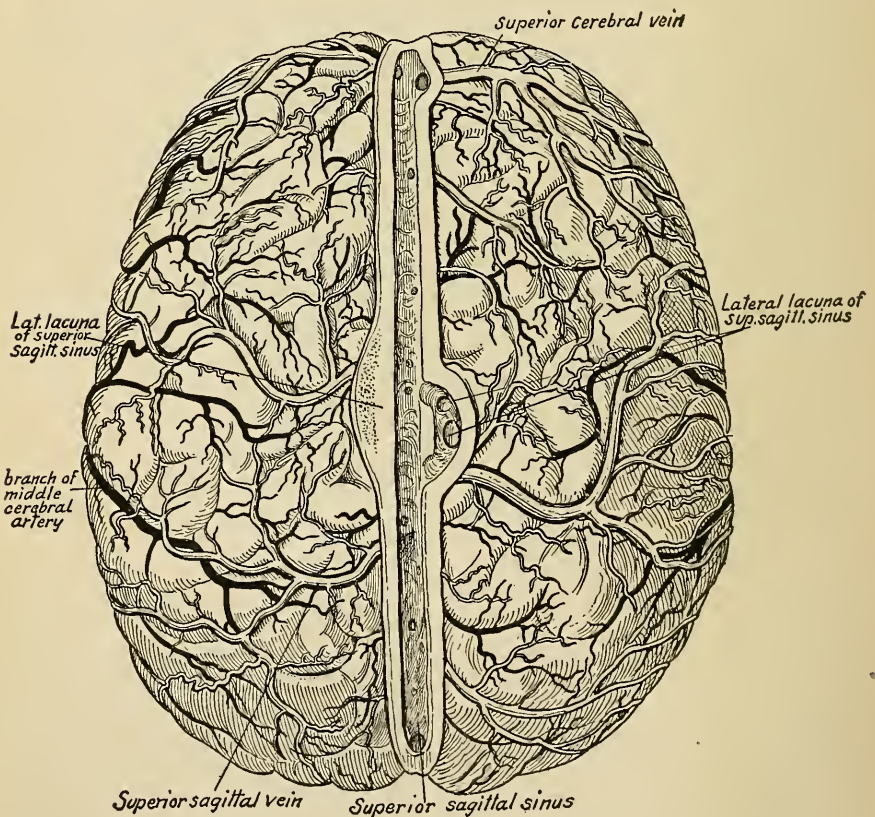

Superior cerebral vein
Lat. lacuna of superior sagitt. sinus
Lateral lacuna of sup. sagitt. sinus
branch of middle cerebral artery
Superior sagittal vein
Superior sagittal sinus

Fig. 61.—The arteries and veins of the brain as seen from above.

of the internal carotid, runs directly backward, and anastomoses with the posterior cerebral and branches of the basilar artery.

The Anterior Choroid Artery.—This is a small branch which arises from the back part of the internal carotid and enters the descending horn of the lateral ventricle. It is distributed to the choroid plexus and to structures in this portion of the lateral ventricle.

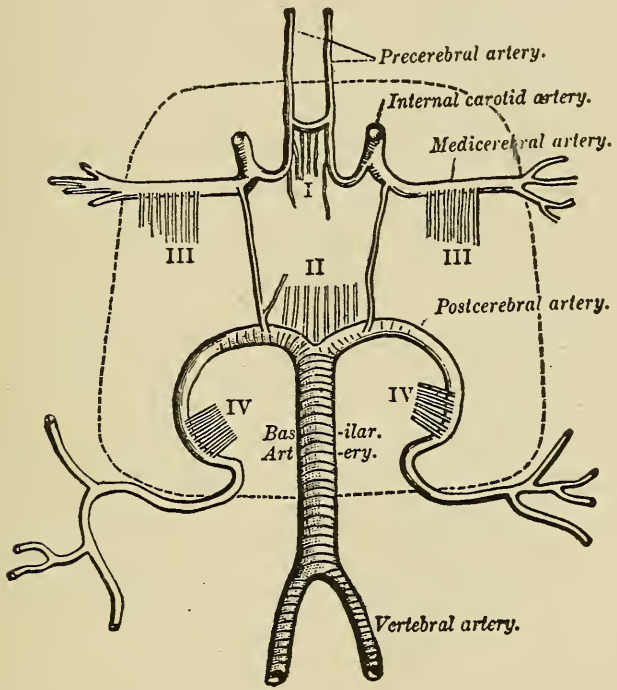


Fig. 62.—Diagram of the arterial circulation at the base of the brain: I, Anteromedian group of ganglion branches; II, posteromedian group; III, right and left anterolateral group; IV, right and left postlateral group. The dotted line shows the limit of the ganglionic circle. (After Charcot.)

The Circle of Willis.—This is the designation by which the remarkable anastomosis at the base of the brain is familiarly known (Fig. 62). It is formed, in front, by the anterior cerebral arteries and branches of the internal carotid, which are connected by the anterior communicating; behind, by the two posterior cerebral branches of the basilar arteries, which are connected on each side with the internal carotid by the posterior communicating artery. From the circle of Willis three trunks arise which,

together, supply each cerebral hemisphere. From the anterior part there are two anterior cerebral; from the anterolateral part, the middle cerebral; and from the posterior part, the posterior cerebral. The principal arteries give origin to two very different systems of secondary vessels. One of these systems has been named the central ganglionic system. The vessels belonging to this system supply the central ganglia of the brain. The other has been named the cortical arterial system: its vessels ramify in the pia mater and supply the cortex and subjacent medullary matter. These two systems, though having a common origin, do not communicate at any point of their peripheral distribution. Between these two systems a borderline of tissue exists where softening is especially likely to occur.

The Central Ganglionic System.—All the vessels belonging to this system are given off from the circle of Willis or from the vessels in its immediate vicinity. These vessels are smaller than those of the cortical system, and are terminal arteries; that is, vessels that, from their origin to their termination, neither supply nor receive any anastomotic branches.

The Cortical Arterial System.—The vessels forming this system are branches of the anterior, middle, and posterior cerebral arteries. They divide and ramify in the substance of the pia mater, and then give off terminal arteries, some of which are short and others long. Each of these terminal branches supplies a definite area of cerebral substance.

FUNCTIONS OF THE WHITE SUBSTANCE OF THE CEREBRUM

Four distinct sets of fiber tracts may be distinguished in the brain: tangential, commissural, associative, and projection.

The *tangential tracts* consist of numerous short fibers, particularly well marked in the cortex, and connecting cells situated within a short distance of one another.

The Commissural Tracts.—These are the pathways that connect similarly situated areas of the two cerebral hemispheres with each other. The corpus callosum is the best developed of any of these pathways. (See Figs. 53 and 54.)

The *associative tracts* are those that interconnect various regions of the brain with each other. (See Fig. 55.)

Projection Tracts.—These are the tracts that connect the cerebral cortex with the parts below. (See Fig. 56.)

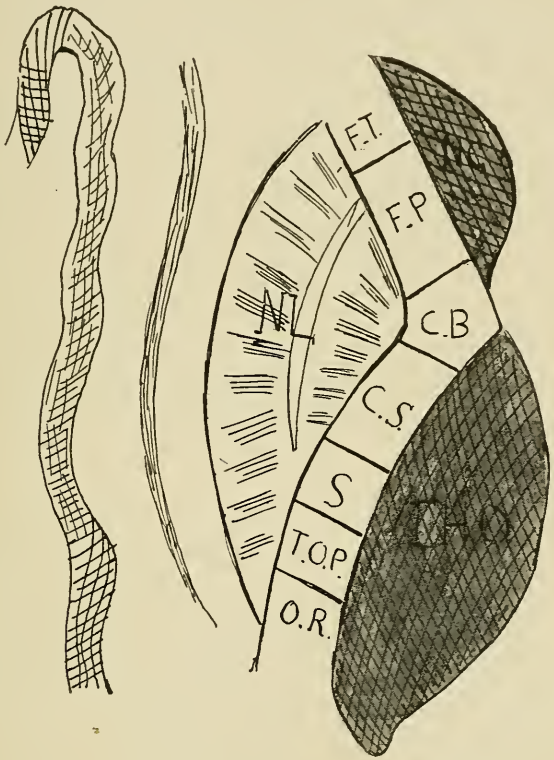


Fig. 63.—Diagram showing relative position of chief tract in internal capsule: *F.T.*, Frontothalamic; *F.P.*, frontopontine; *C.B.*, corticobulbar; *C.S.*, corticospinal; *S.*, tegmental sensory; *T.O.P.*, temporo-occipitopontine; *O.R.*, optic radiation; *N.L.*, lenticular nucleus; *N.C.*, caudate nucleus; *T.H.O.*, optic thalamus.

The region between the optic thalamus and the caudate nucleus internally, and the lenticular nucleus externally, is called the internal capsule. (See Fig. 57.)

The projection tracts from the frontal lobe occupy the anterior limb of the internal capsule. The projection tracts from the third frontal and lower precentral convolution occupy the knee of the internal capsule. Posterior to these are the projection

tracts from the middle portion of the precentral convolution, and posterior to these are the projection tracts from the superior portion of the precentral convolution. The posterior third of the posterior limb of the internal capsule is occupied by fibers that have ascended from the spinal cord, medulla, lenticular nucleus, and optic thalmus, to end around cells in the parietal and temporal lobes.

The knee and the anterior two-thirds of the posterior limb of the internal capsule are occupied by fibers that transmit impulses which result in voluntary motion. The posterior third of the posterior limb is occupied by fibers that transmit impulses which bring about sensory impressions (Fig. 63).

In the knee and posterior limb of the internal capsule, from before backward, are placed, in the order in which they are named, the pathways of the stimuli which bring about movements of the tongue, lips, face, eyes, hands, arms, legs, and trunk. In the posterior third are the pathways of the stimuli which are reflected on consciousness as sensation.

FUNCTION OF THE CEREBRAL CORTEX

In the cells of the first and frontal lobes are centered the memories, which may be either inherited or acquired, and which exercise supervision over the moral character and intellectual abilities of the individual. The third frontal convolution on the left side, in right-handed individuals, and on the right side, in left-handed individuals, is the motor speech center. Here are stored the memories which, when called into activity, result in the sounds which we understand as language. In the posterior part of the frontal lobe, the precentral convolution, and most likely extending for a short distance posterior to the fissure of Rolando in the postcentral convolution, are situated the centers which control the voluntary movements of the body. Those in the left cerebral cortex supervise the motor activities of the right side, and those in the right cerebral cortex govern the motor activities of the left side. The centers for the voluntary movements of the eyes and face are situated lowest down and somewhat anteriorly in the precentral convolution; those for the

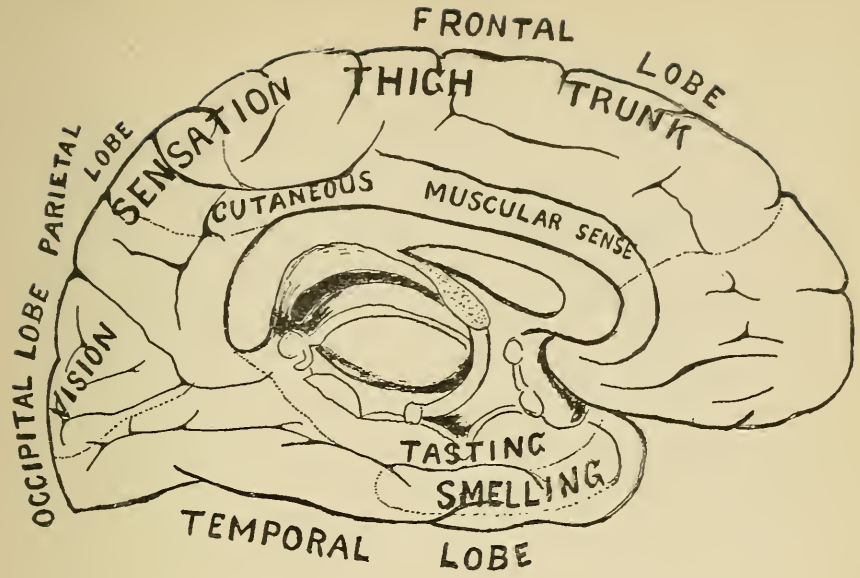


Fig. 63 *a*.—Median view of the cerebral cortex showing location of important centers (diagrammatic).

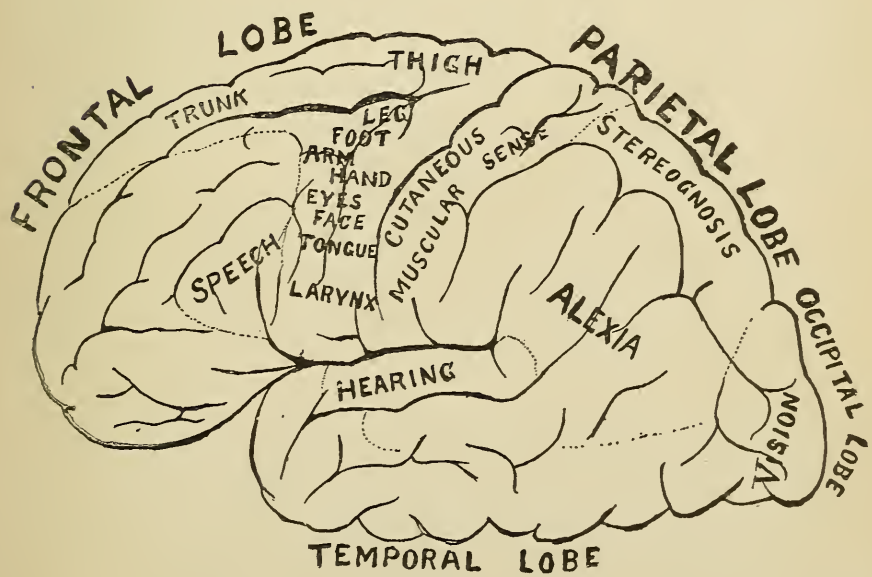


Fig. 63 *b*.—Lateral view of the cerebrum showing the location of important centers (diagrammatic).

movements of the fingers, hands, and arms being somewhat higher and farther back. Above these are the centers for the movements of the trunk and legs.

In the postcentral convolution and in the anterior part of the parietal lobes the stimuli that are reflected upon consciousness as sensation are received.

In the angular gyrus are associated various impressions that result in the stereognostic sense. In the cuneus and on the lateral aspect of the occipital lobe are the centers of sight, and in these centers are stored the memories that enable us to recognize the things that are seen. In the anterior part of the lateral aspect of the occipital lobe is the reading center. In the superior temporal convolution are stored the memories of sounds heard—the auditory sense, which enables an individual to understand spoken language. In the internal aspect of the temporal lobe, in the uncus and hippocampal convolution, are the cells in which are stored the memories of odors—the olfactory sense—and those of taste—the gustatory sense.

The Functions of the Basilar Ganglia.—The corpora striata and the optic thalami are believed to be intermediate stations for the conduction of sensation. The optic thalami also receive filaments from the optic tracts, and are intermediate stations of vision and of reflex activity.

The functions of about one-third of the cortex of the brain are understood, and the remainder, or two-thirds, is believed to be concerned with associative interactivity.

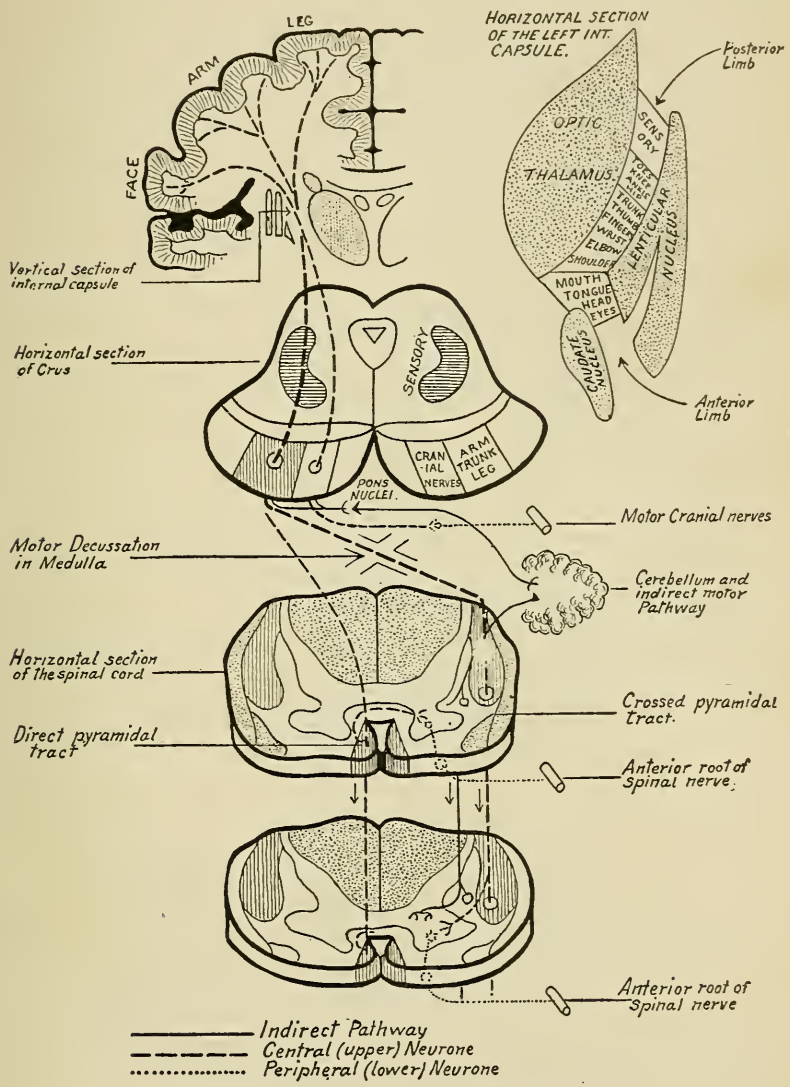
The Functions of the Hypophysis Cerebri.—The anterior lobe secretes a substance that enters the blood and promotes skeletal growth. The secretion of the posterior lobe influences the processes of tissue metabolism and the activity of the renal and vascular systems.

The Course of a Motor and a Sensory Impulse From the Point of its Origin to the Area in which it Produces its Effect.—A *motor impulse* originating in the precentral convolution descends through the projection pathways into the internal capsule, where it passes through an area somewhere between the knee and the posterior third of the posterior limb. It then crosses the opposite side, to end around the nucleus of the third or fourth nerve, or

it may enter the crura, where it will pass through an area occupying the middle three-fifths of the anterior portion of this structure. It may then cross the opposite side, to end around the motor nucleus of the fifth nerve, or it may enter the pons, where it will pass through an area situated anteriorly. It may next cross the opposite side, and end around the nucleus of the sixth or seventh nerve, or it may descend into the pyramids of the medulla, and at this point cross the opposite side, to stimulate the nucleus of the ninth, tenth, eleventh, or twelfth nerve. Instead, however, it may continue downward through the pyramids of the medulla to the lower portion of the medulla, where 90 per cent. of the fibers cross the opposite side and make up the crossed pyramidal tracts, 10 per cent. descending on the same side as the direct pyramidal tracts of the spinal cord. As the impulse descends through the crossed pyramidal tracts, it will stimulate to activity cells in the anterior horns of the same side of the cord as that on which it descends. If it descends in the direct pyramidal tract, before it stimulates cells in the anterior horn it will cross in the anterior or white commissure of the spinal cord, to stimulate cells in the anterior horns of the opposite side of the cord. As the result of the stimulation of the cells of the nuclei of the cranial nerve, or of cells in the anterior horn of the spinal cord, new impulses arise which are transmitted through the cranial nerves or spinal nerves, to stimulate the muscle-fibers to activity and result in the execution of purposive and voluntary actions (Figs. 64 and 65; also see Figs. 33 and 67).

The Course of a Sensory Impulse.—The stimulus, originating in the periphery, affecting the termination of a sensory nerve travels upward through the ganglionic cells which are situated on all sensory nerves; it then enters the spinal cord through the posterior roots or the medulla or pons, through the superficial origin of the cranial nerves. If the sensory impulse enters the medulla or the pons, it stimulates to activity cells situated beneath the floor of the fourth ventricle. From these cells new fibers arise which transmit the impulse. These fibers ascend on the same or on the opposite side through the fillet, to end around the cells in the lenticular nucleus or optic thalami.

Some fibers ascend through the posterior portion of the posterior limb of the internal capsule, to end around the cells in the



Figs. 64 and 65.—Motor pathway.

parietal lobe. A sensory impulse may also be conducted by other fibers arising in cells beneath the floor of the fourth ventricle, which may or may not cross the midline, and which may

end around cells in the pons, the medulla, the crura, or the vermis of the cerebellum (Fig. 66; also see Fig. 33 and Fig. 67).

If the impulse is transmitted by one of the spinal nerves,

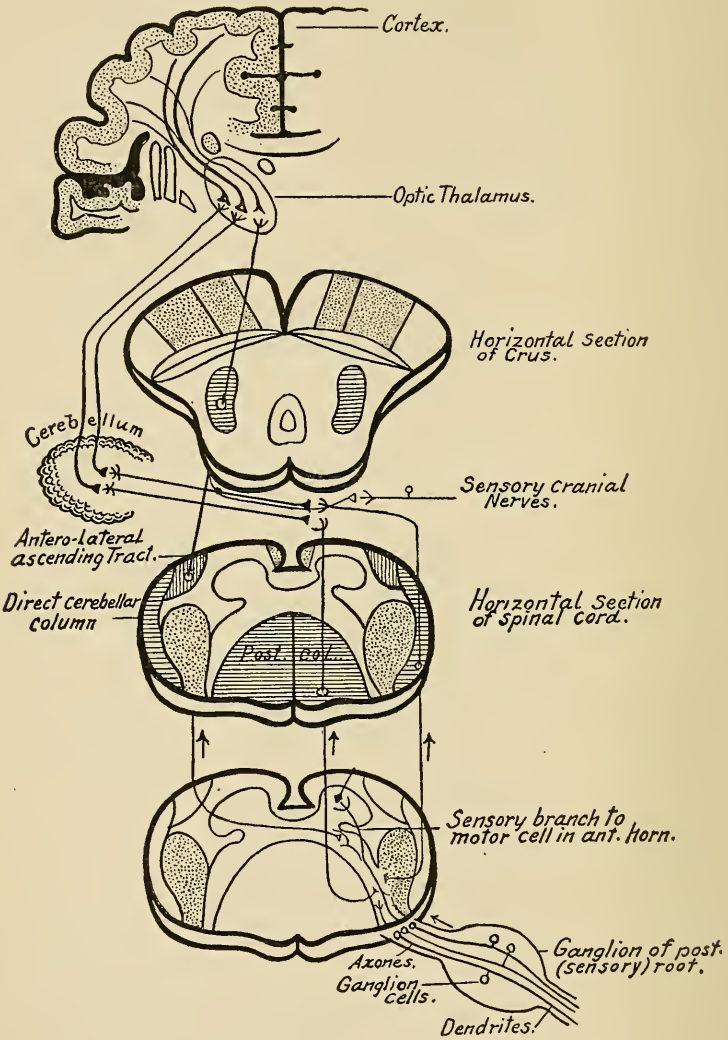


Fig. 66.—Sensory pathways.

it enters the spinal cord through the posterior roots, where it may pursue one of the several pathways. (See Fig. 33.)

Muscle Sense.—A muscle sensation is transmitted by fibers

that ascend a short distance in Lissauer's column and then enter the column of Goll or Burdach. Most of these fibers are on the same side and a few are on the opposite side. The fibers ascend and end around cells in the nucleus of Goll or Burdach. From these cells fibers arise that cross to the opposite side, and ascend in the fillet through the medulla, pons, or crura. Some fibers end around cells in the lenticular nucleus or optic thalami, and from these cells new fibers arise which enter the posterior part of the internal capsule, to terminate around cells in the parietal lobe. Other fibers originating in cells in the nucleus of Goll and Burdach may continue to ascend through the medulla, pons, and crura, to the posterior part of the internal capsule, to end around cells in the parietal lobe. Still other fibers (arciform fibers), originating in cells situated in the nucleus of Goll or Burdach, most of which cross the midline, cross through the reticular formation and enter the inferior peduncle of the cerebellum, to end around cells in the vermis.

Pain and Temperature Sense.—The stimuli which originate in the periphery ascend through the sensory nerves, the ganglion on the posterior roots; they ascend through the posterior roots to enter the spinal cord. From here they ascend for a short distance in Lissauer's column, after which they enter the posterior horns to stimulate cells in the anterior part of the posterior horns. From these cells fibers arise that ascend and cross through the gray commissure to enter the anterolateral column of the opposite side. The impulse then ascends through this column, which becomes deflected dorsalward and centrally upon entering the medulla, where it ascends through the reticular formation, becoming a part of the lemniscus of the medulla and pons and entering the tegmentum of the crura. Some of these fibers terminate around cells in the optic thalami and lenticular nucleus, the greater number of them ascending through the posterior limb of the internal capsule to enter the parietal lobe, where they end around cells in the cortex of the lobe.

Tactile Sense.—This form of sensation is transmitted through two pathways: it partly follows the pathway that conducts muscle sensations, and in part follows the pathway that conducts pain and temperature sense.

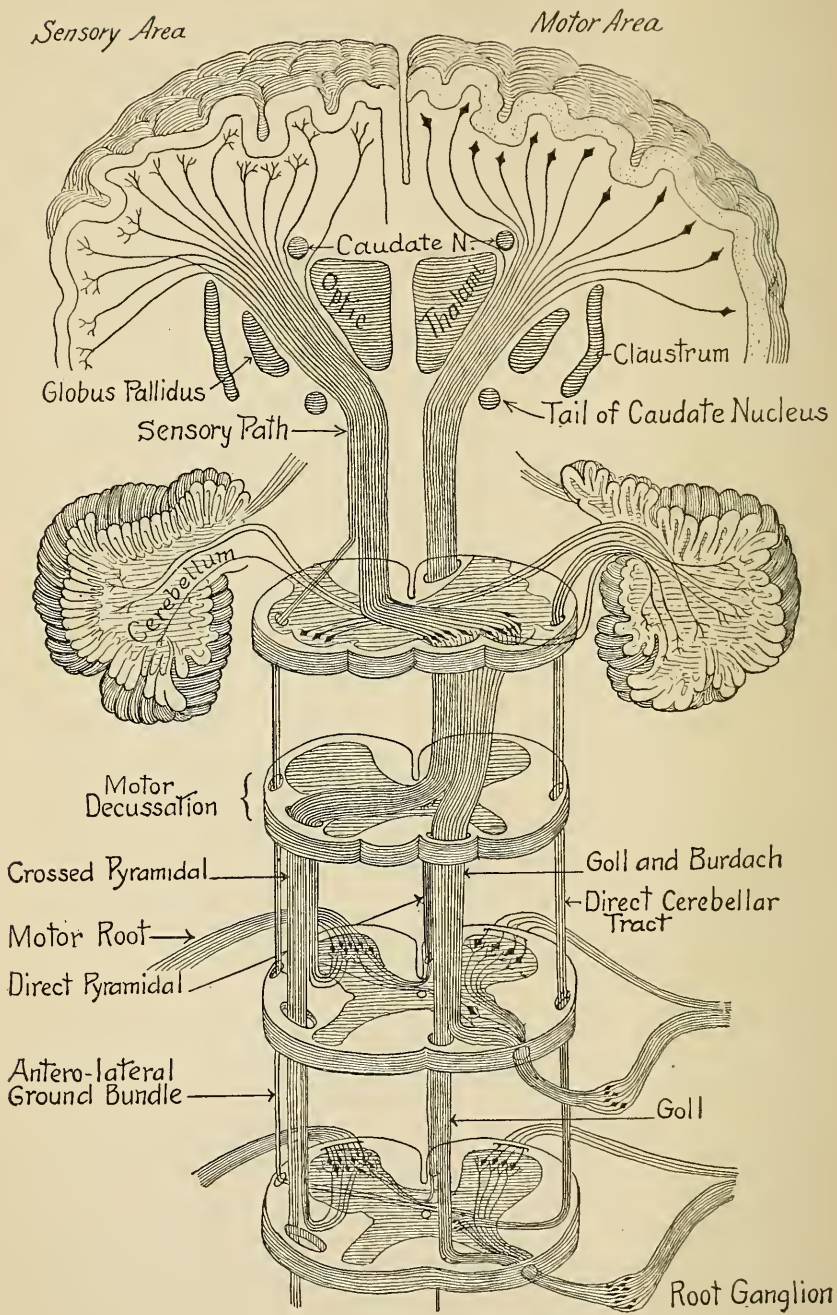


Fig. 67.—Combined motor and sensory pathways.

Equilibrium.—The principal pathway for this function of the nervous system is the direct cerebellar tract. Stimuli which assists in maintaining this function originate in the periphery, ascend through the peripheral nerves, through the ganglia on the posterior roots, enter the cord through the posterior roots, then ascend a short distance in Lissauer's marginal zone, then enter the posterior horns where they stimulate to activity cells in the column of Clark. These stimuli are then transmitted through fibers which arise from the cells in the column of Clark and which cross through the gray and white matter of the posterolateral portion of the cord, then turn upward and form the direct cerebellar tract; this column ascends in the posterolateral margin of the cord; in the medulla it is deflected slightly forward, forms an important portion of the inferior cerebellar peduncle, its fibers entering the cerebellum and terminating around cells in the vermis.

Impulses which assist in maintaining equilibrium also originate in the spiral ganglia and are transmitted through the vestibular portion of the eighth nerve to the medulla; from here, through the interposition of other neurons, they are transmitted to the vermis of the cerebellum.

A careful study of Figs. 33 and 37, showing the pathways of these various forms of sensation, will disclose the relative spatial relation of these pathways to one another, to the cellular constituents of the spinal cord, to the nuclei of the cranial nerves, and to the tracts of the medulla, pons, and crura.

ANATOMY AND PHYSIOLOGY OF THE CRANIAL NERVES

The First or Olfactory Nerve.—This nerve receives stimuli at its special endings in the Schneiderian mucous membrane of the nose. The fibers of this nerve enter the cranial cavity through the cribriform plate of the ethmoid bone, and there enter the cells of the olfactory bulbs. The axons of these cells pass through the olfactory tracts. The impulse then enters either the medial or the lateral root, the medial root going in part to the callosal and subcallosal gyrus, and in part to the area of Broca. The

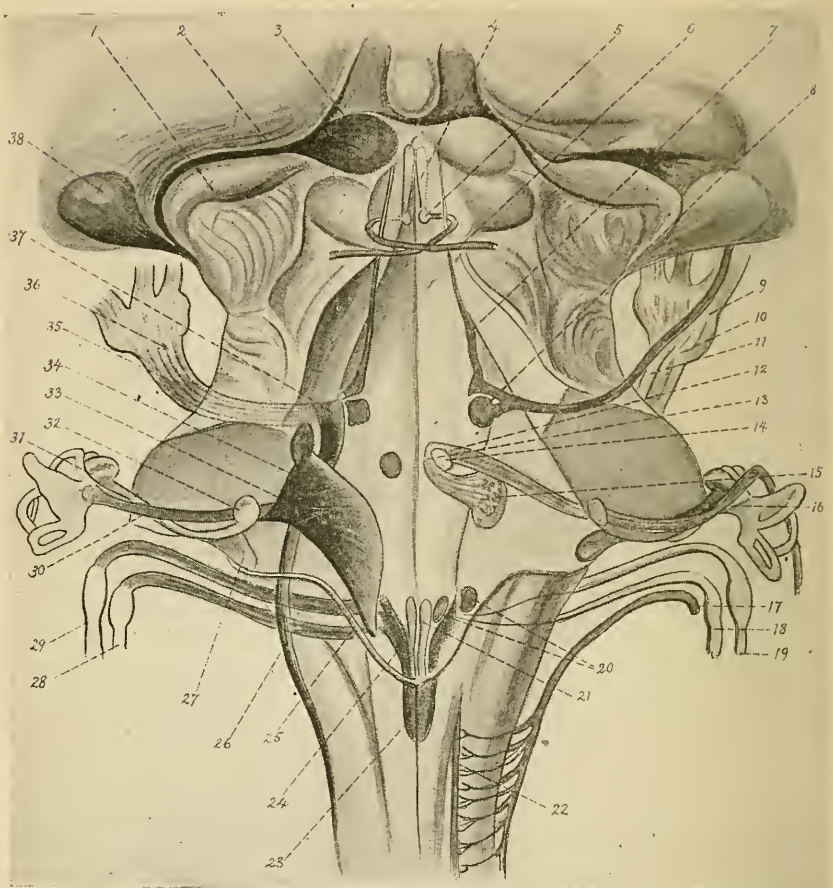


Fig. 68.—Nuclei of origin of motor and primary terminal sensory nuclei of cerebral nerves (Held), schematically represented in a supposedly transparent brain stem viewed from behind: 4, Nucleus of the third nerve (n. oculomotorii); 5, nucleus of the fourth nerve (n. trochlearis); 6, the fourth nerve; 7, the descending (motor) root of the fifth nerve; 8, the principal motor nucleus of the fifth nerve; 9, the semilunar ganglion (g. Gasseri); 26, the ascending (sensory) root of the fifth nerve; 14, nucleus of the sixth cranial nerve; 15, nucleus of facial (seventh) nerve; 16, the facial nerve; 34, 33, nucleus of the vestibular branch of the eighth cranial nerve; 32, ventral nucleus of the cochlear branch of the eighth nerve; 27, dorsal nucleus of the cochlear branch of the eighth nerve; 19, 29, the glossopharyngeal nerve; 18, 28, the vagus nerves 20, motor nuclei of vagus and glossopharyngeal (nucleus ambiguus and nucleus dorsalis); 23, 24, nucleus of the alæ cinereæ, the solitary bundle and its nucleus; 17, the eleventh or spinal accessory nerve; 22, nucleus of the spinal accessory; 21, nucleus of the hypoglossal nerve. (From Spalteholz, "Human Anatomy.")

lateral root enters the anterior perforated space, and terminates finally in the uncinate gyrus of the temporal lobe. A few of the fibers of the olfactory tract terminate in the anterior end of the optic thalamus, whereas others pass through the anterior white commissure. The function of this nerve is to receive, in specialized endings and transmit, stimuli that result in the sense of smell.

The Second or Optic Nerve.—The second or optic nerve is the nerve that receives and conducts stimuli resulting in sight. The stimuli affect the specialized terminations of these nerves in the fundus of the eyes. All stimuli originating to the right of the midline affect the left halves of the fundi of the two eyes; those which originate to the left of the midline affect the right halves of the fundi of the two eyes. These stimuli are conducted backward through the optic nerves. Those stimuli which have arisen in the right halves are conducted backward through the right optic tract, whereas those which affect the left halves are conducted backward through the left optic tract. The optic tracts send fibers into the external geniculate body and the optic thalamus of the same side. In the optic thalamus new fibers arise—the optic radiations—which appear posterior to the internal capsule and end in the cuneus of the occipital lobe.

All objects to the left of the midline are seen as the result of stimulation of the right occipital cortex, and that all objects to the right of the midline are seen as the result of stimulation of the left occipital cortex.

The Third or Oculomotor Nerve.—This is the motor nerve of all the external and internal muscles of the eye except the sphincter palpebra, the external rectus, and the superior oblique. Its nucleus of origin is in the gray matter around the aqueduct of Sylvius, in the lower portion of the crura, and consists of a long column of cells (Fig. 69). The nerves, which most likely contain filaments from both nuclei, pierce the structures of the crura and appear one on each side of the midline, on the inner side of the cerebral peduncles, immediately above the pons. The nerve pursues an anterior course, and pierces the dura mater in front of and external to the posterior clinoid process. It passes along the outer wall of the cavernous

sinus above the other orbital nerves, receiving in its course filaments from the cavernous plexus of the sympathetic and from the ophthalmic division of the fifth nerve. It enters the orbit through the sphenoidal fissure. It divides into numerous

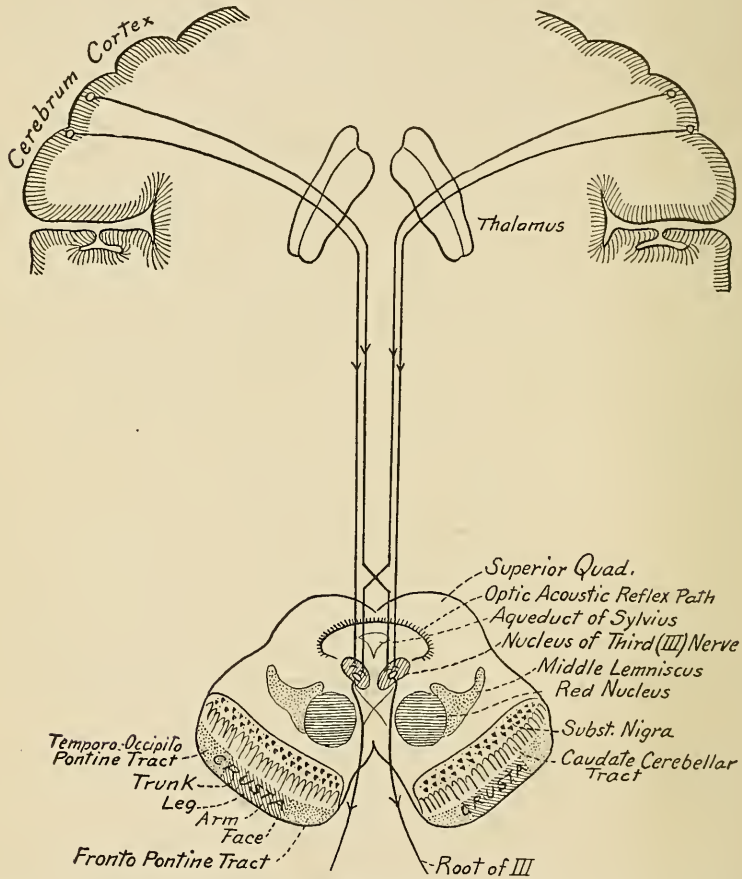


Fig. 69.—Diagram showing the deep and superficial origin of the third nerves, the relation of these nerves to the crura cerebri, and their connection with the cerebral cortex.

branches, supplying the superior rectus, inferior rectus, levator palpebræ, internal rectus, and inferior oblique, sending a branch also to the lenticular ganglion, and from this, through a short ciliary nerve, to the ciliary muscle and sphincter of the pupil.

The Fourth or Trochlear Nerve (Fig. 70).—The nucleus of origin of this nerve is immediately below that of the third nerve. Its fibers take a backward course and cross in the midline in the valve of Vieussens. It appears to the outer side of the cerebral peduncles, between the cerebrum and cerebellum, and then pursues an anterior course with the third nerve, to enter the orbit through the sphenoidal fissure to supply the superior oblique muscle.

The **fifth** or **trifacial nerve** is the largest of the cranial nerves (Fig. 71). It resembles the spinal nerve in that it arises by two

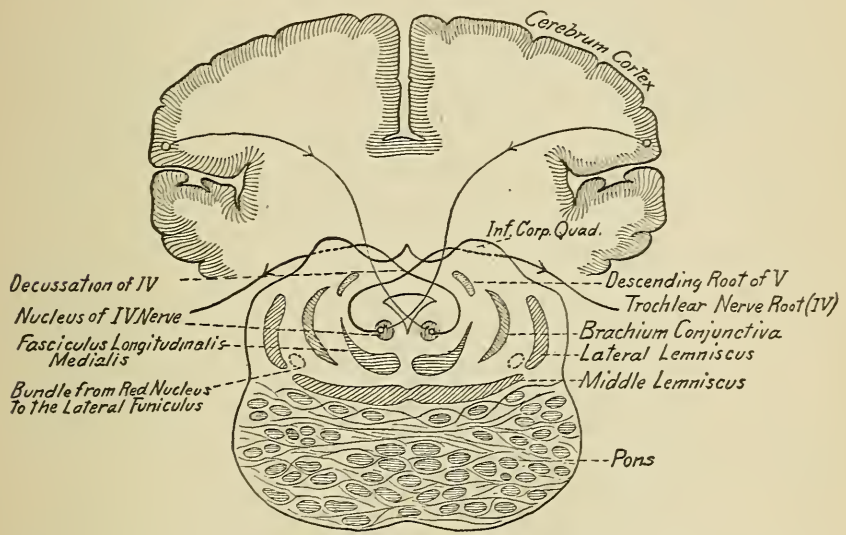


Fig. 70.—Diagram showing the deep and superficial origin of the fourth nerves, the relation of these nerves to the crura cerebri, and their connection with the cerebral cortex.

roots and is a mixed nerve. Its motor root arises from cells lying below the floor of the fourth ventricle, near the lateral angle. Its sensory root has its cells of origin in the Gasserian ganglion. The motor root pierces the structures of the pons and appears at the side near its upper margin. The sensory root enters the pons at this point, dividing into ascending and descending fibers that end around cells beneath the floor of the fourth ventricle, extending upward as far as the crura and downward as far as the second or third cervical segment of the spinal cord. The

two roots of the fifth nerve pass forward through an oval opening in the dura mater at the superior border of the petrous portion of the temporal bone, above the internal auditory meatus. They then pursue a course forward to the apex of the petrous portion of the temporal bone. Here is situated the Gasserian ganglion, under which the motor root passes without entering

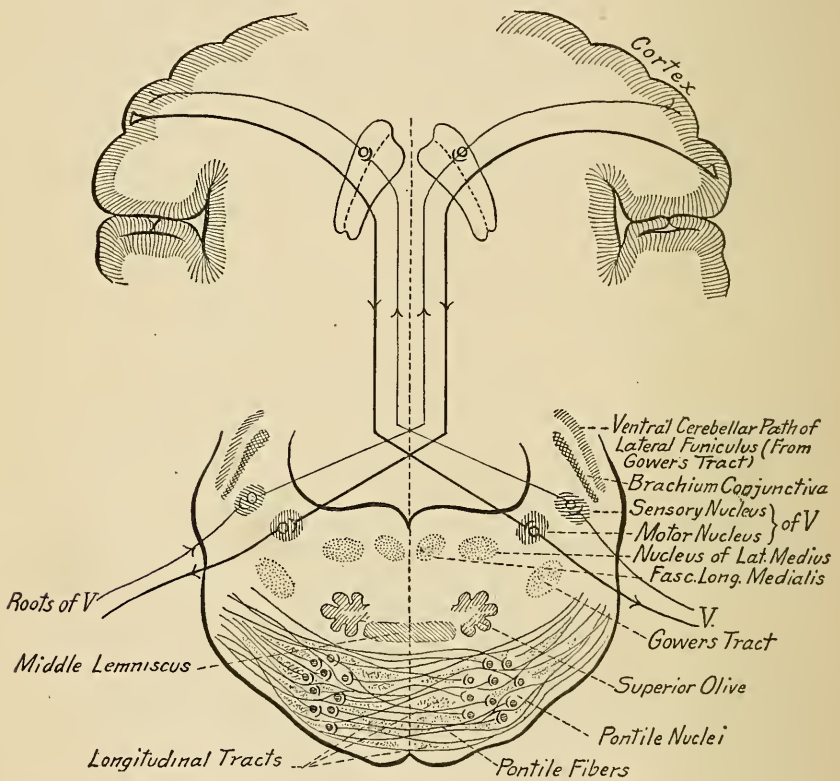


Fig. 71.—Diagram showing schematically the connection of the fifth nerve with the cerebral cortex and the relationship of the nuclei of this nerve to the structures of the pons.

into it. The Gasserian ganglion is covered by the dura mater, and receives branches from the carotid plexus of the sympathetic nerve. It gives off the following branches: The *ophthalmic*, a short branch, which passes forward along the outer wall of the cavernous sinus, entering the orbit through the sphenoidal fissure (Fig. 72). The *superior maxillary*, which passes

forward, emerges from the cranial cavity through the foramen rotundum, and then crosses the sphenomaxillary fossa, entering the infra-orbital canal in the floor of the orbit and appearing upon the face at the infra-orbital foramen. The *inferior maxillary nerve*, which consists of two portions—a large sensory portion, being the third branch of the Gasserian ganglion, and a small portion, which is the motor portion of the fifth nerve and which has passed beneath the ganglion. These two portions emerge

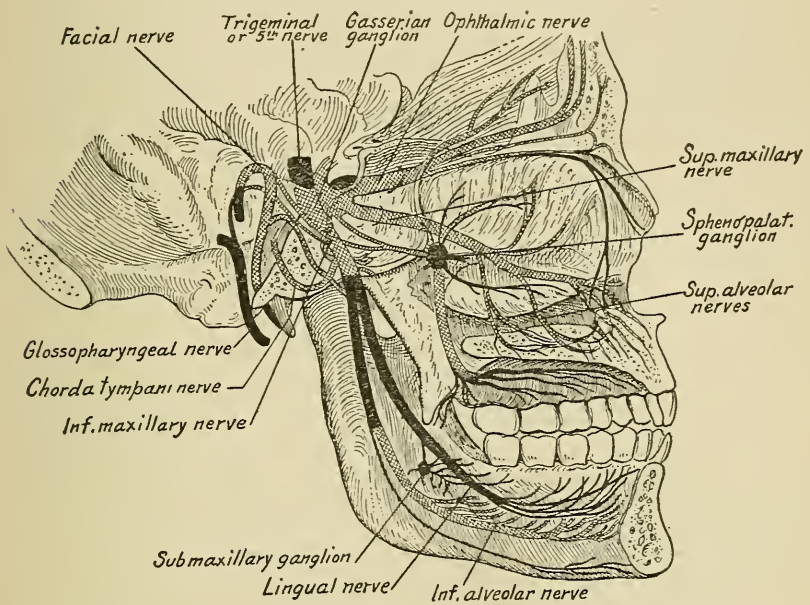


Fig. 72.—The trigeminal nerve and its communications with the facial and glossopharyngeal nerves. (After Sobotta.)

from the cranial cavity through the foramen ovale, where they fuse with each other.

This nerve supplies sensation to the anterior part of the head, the face, the orbit, the structures within the orbit, the nose, the mouth, and perhaps the upper portion of the neck. Its motor portion is distributed to the muscles of mastication.

The **sixth** or **abducent nerve** supplies the external rectus muscle of the eye. Its nucleus of origin is situated beneath the floor of the fourth ventricle, in front of the acoustic striæ. Its

fibers pierce the structures of the medulla and appear beneath the pons near the midline. The nerve runs almost directly forward, crosses the pons, lying to the outer side of the internal carotid artery, and enters the orbit through the sphenoidal

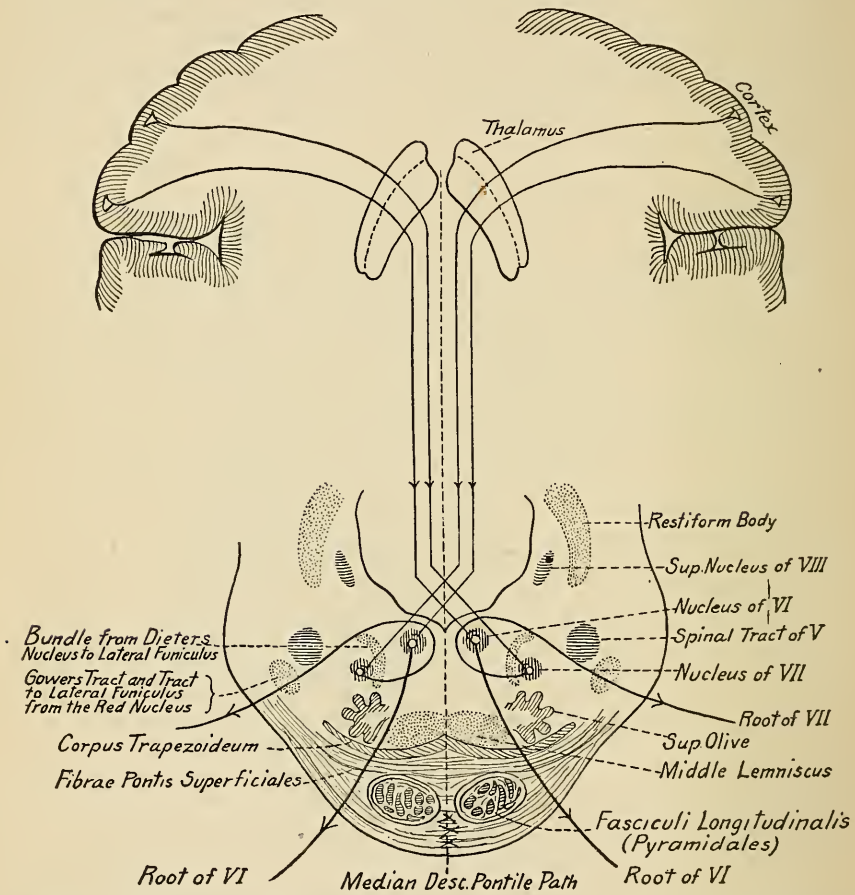


Fig. 73.—Transverse section of the medulla at the level of the sixth and seventh nerves, showing schematically the connection of the nuclei of these nerves with the cerebral cortex and their relation to each other and to the structures of the upper medulla.

fissure. It pursues the longest intracranial course of any of the cranial nerves (Fig. 73).

The **seventh** or **facial nerve** is distributed to the muscles of expression, to the platysma, buccinator, posterior belly

of the digastric, stylohyoid muscles, etc. Through its chorda tympani branch it supplies the lingualis muscle, and through its tympanic branch, the stapedius. Its nucleus of origin is beneath the floor of the fourth ventricle, anterior and external to the nucleus of the sixth nerve. On leaving the nucleus the fibers are at first directed backward and inward toward the middle line; they then ascend for a short distance behind the nucleus of the sixth nerve, close to the floor of the fourth ventricle. They next bend down and outward over the upper end of the nucleus of the sixth, and then are directed anteriorly outward and downward, appearing beneath the lower border of the pons in the groove between the olivary and restiform bodies. The nerve then passes forward and outward in close apposition with the eighth, and enters the internal auditory meatus. At the bottom of the meatus the facial nerve enters the aqueductus Fallopii, and follows the course of that canal through the petrous portion of the temporal bone, emerging at the stylomastoid foramen. From this aperture it runs forward in the substance of the parotid gland, crosses the external carotid artery, and divides into branches that are distributed to the side of the head, face, and upper part of the neck, and supplying the superficial muscles in this region.

On the facial nerve, just within the internal auditory meatus, is the *geniculate ganglion*. The proximal root, called the pars intermedia, or the nerve of Wrisberg, enters the medulla between the facial and the auditory nerve, and probably ends around cells in the glossopharyngeal nucleus. The peripheral root of this ganglion joins the seventh, a portion of its fibers leaving the seventh beneath the floor of the middle ear as the chorda tympani nerve, to join the inferior maxillary branch of the fifth. Some of its fibers probably serve for the conduction of sensation from a small cutaneous area and the external auditory canal (Fig. 74).

The fibers of the chorda tympani, which conduct the sense of taste from the anterior two-thirds of the tongue, after entering the geniculate ganglion, may transmit stimuli through the pars intermedia to the glossopharyngeal nucleus; or the stimuli may be transmitted from the geniculate ganglion through the great superficial petrosal nerve to Meckel's ganglion, thence to the

superior maxillary division of the fifth nerve, and through this nerve to the cortical taste-centers in the temporal lobe.

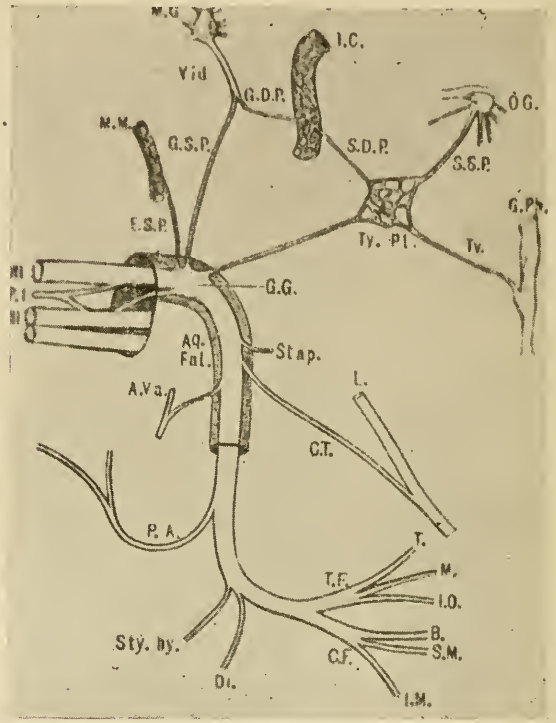


Fig. 74.—The facial nerve with its branches and communications in the aqueduct of Fallopius: VII., Facial nerve; *P.I.*, pars intermedia; VIII., auditory nerve; *Aq.Fal.*, aqueduct of Fallopius; *G.G.*, geniculate ganglion; *E.S.P.*, great superficial petrosal nerve; *G.P.D.*, great deep petrosal nerve; *I.C.*, internal carotid artery; *Vid.*, vidian nerve; *M.G.*, Meckel's ganglion; *Ty.Pl.*, tympanic plexus; *S.D.P.*, small deep petrosal nerve; *G.Ph.*, glossopharyngeal nerve; *Ty.*, tympanic branch; *S.S.P.*, small superficial petrosal nerve; *O.G.*, otic ganglion; Stap, nerve to stapedius; *C.T.*, chorda tympani nerve; *L.*, lingual nerve; *A.Va.*, communication with auricular branch of vagus; *P.A.*, posterior auricular nerve; *Sty. hy.*, nerve to stylohyoid; *Di.*, nerve to digastric (posterior belly); *T.F.*, temporofacial division; *C.F.*, cervicofacial division; *T.*, temporal; *M.*, malar; *I.O.*, infra-orbital; *B.*, buccal; *S.M.*, supramandibular; *I.M.*, inframandibular branches.

The Eighth or Auditory Nerve.—This is the special nerve of the sense of hearing, being distributed exclusively to the internal ear. Its cells of origin are in the vestibular and spiral ganglia. These are situated respectively in the semicircular canals and the

internal ear. The nerve, arising from these ganglia, emerges from the petrous portion of the temporal bone at the internal auditory meatus, where it is in close apposition to, and remains connected with, the seventh nerve, entering the medulla in the same groove with but behind the facial.

The *medial root*, which is the *vestibular division*, passes backward through the substance of the medulla and pons, internal to the restiform bodies. The fibers of this root divide into ascending and descending branches. The ascending branches end around cells beneath the floor of the fourth ventricle, from which fibers pass to the cerebellum. The descending branch also ends around cells beneath the floor of the fourth ventricle, and some of these fibers can be traced through the medulla to the spinal cord as the vestibulospinal tracts. This root is connected principally with the cerebellum, the optic thalami, and the nucleus of the sixth, third, fourth, and fifth nerves. It conducts impulses that have to do with the sense of equilibration.

The *lateral root*, or *cochlear division*, is the true nerve of hearing. It arises as the central processes of the bipolar cells in the spiral ganglion of the cochlea. It passes backward into the medulla, on the outer side of the restiform body. Its fibers divide into ascending and descending branches. The ascending branches end around cells of the ventral cochlear nucleus. The descending branches end around cells in the dorsal cochlear nucleus. The further course of auditory impulses is from the ventral nucleus through the trapezoid body; from the dorsal nucleus through the acoustic striæ and the trapezoid body, to join the superior olives; through the lateral fillet of the same and opposite side to the posterior corpora quadrigemina, to the internal geniculate body; through the posterior part of the internal capsule, to the superior temporal convolution.

The **ninth** or **glossopharyngeal** is a mixed nerve, having a small motor root in common with the tenth nerve. The origin of the motor root is from the cells of the nucleus ambiguus, which is situated beneath the floor of the fourth ventricle. The sensory root has its origin in cells of the petrous and jugular ganglia. The motor root emerges from the medulla in the groove between the olivary and restiform bodies (Fig. 75). The

sensory root enters this groove, dividing into ascending and descending branches that end around cells beneath the floor of

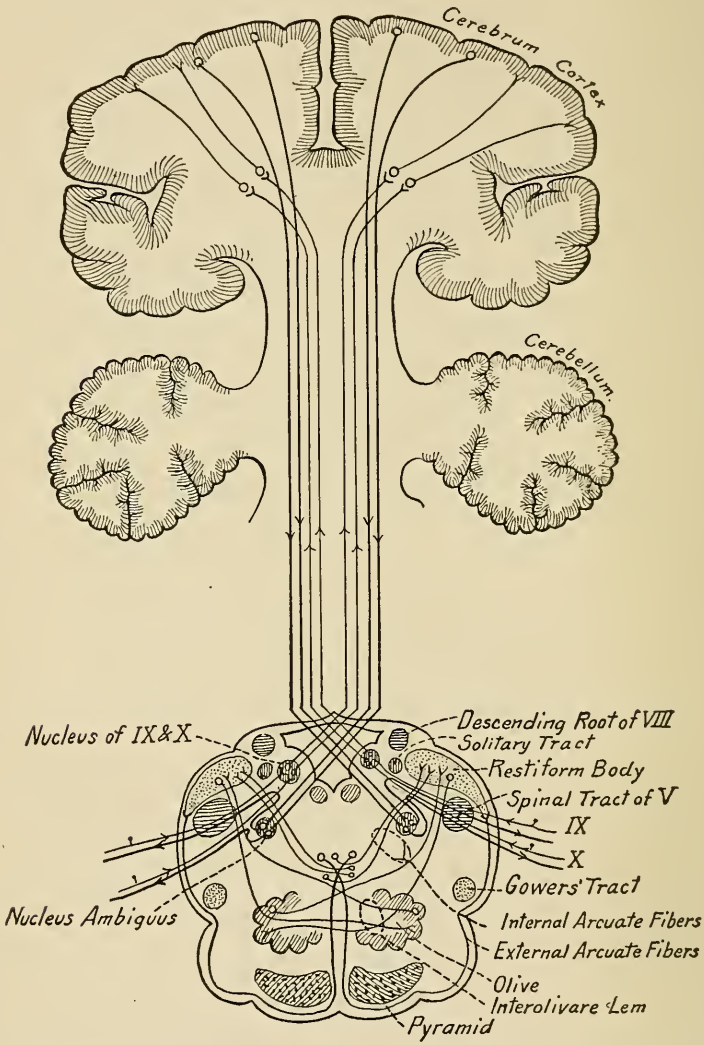


Fig. 75.—Transverse section of the medulla at the level of the ninth and tenth cranial nerves, schematically showing the relation of the nuclei of these nerves to the structures of the medulla and their connection with the cerebral cortex.

the fourth ventricle. From its superficial origin it passes downward and leaves the skull at the central part of the jugular

foramen. In this foramen the nerve presents two enlargements —the superior, called the jugular ganglion, and the inferior and larger, the petrous ganglion.

This nerve is distributed to the tongue and pharynx, being the nerve of sensation to the mucous membrane of the pharynx and fauces. It is a special nerve of taste, being distributed to the posterior part of the tongue.

The **tenth** or **vagus** or **pneumogastric nerve** is a mixed nerve, its motor root originating in cells of the nucleus ambiguus in common with that of the ninth nerve. Its sensory root arises in two ganglionic enlargements on the front of this nerve—the ganglion of the root and the ganglion of the trunk. The motor root emerges and the sensory root enters the groove between the olivary and restiform bodies, below the ninth. The nerve passes outward and downward to the jugular foramen, through which it emerges from the cranium. In this foramen the nerve presents its two ganglionic enlargements.

The tenth nerve has a wider distribution than any of the other cranial nerves. Passing through the neck and thorax to the upper part of the abdomen, it supplies the organs of speech and respiration with motor and sensory fibers, and the pharynx, esophagus, stomach, and heart with motor fibers. The nerve communicates freely with the ninth, the eleventh, and the sympathetic ganglia in the neck, thorax, and upper abdomen.

The Eleventh or Spinal Accessory Nerve.—This nerve consists of two parts—a spinal portion and an accessory portion. The spinal portion originates from cells in the spinal cord, situated in the lateral horn of the gray matter, and as low down as the fifth cervical segment. These fibers appear on the lateral aspect of the cord, behind the anterior root, and form a trunk that ascends and enters the skull through the foramen magnum. It is then directed outward to the jugular foramen, where it joins the accessory portion. The accessory portion originates in a group of cells situated beneath the floor of the fourth ventricle, above and to the side of the nucleus of the twelfth nerve. It emerges from the medulla in the groove between the olivary and restiform body, below the tenth nerve, entering the jugular foramen, where it joins the spinal portion. It emerges from the

cranial cavity through the jugular foramen, descends in front and to the side of the spinal column, giving off branches to the trapezius and to the sternocleidomastoid muscle. It also sends filaments to the second and third cervical nerves, and assists in the formation of the cervical plexus. (See Figs. 26, 27, 32, 33.)

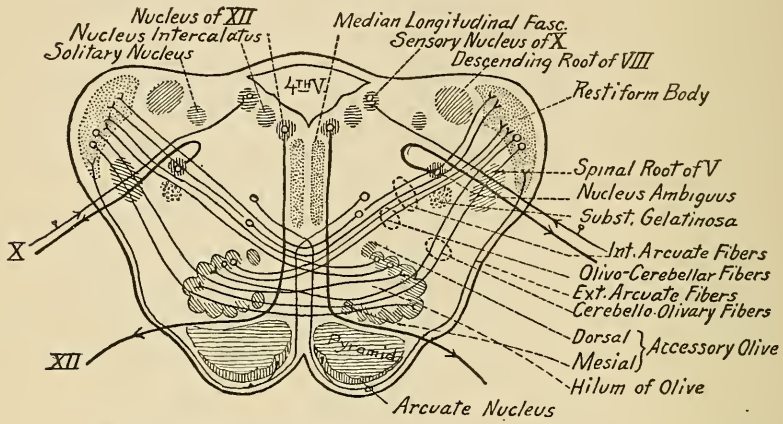


Fig. 76.—Transverse section of the medulla about the middle of the olive, schematically showing the nuclei of the ninth, tenth, and eleventh nerves and their relation to the structures of the medulla (Spalteholz).

The **twelfth** or **hypoglossal nerve** originates from a group of cells lying near the middle line, close to the surface, in the lower half of the floor of the fourth ventricle. Its fibers pierce the reticular formation, and appear in the groove between the pyramid and the olivary body. This nerve emerges from the cranial cavity through the anterior condyloid foramen, and is distributed to the muscles of the tongue. It sends branches to the sternothyroid, sternohyoid, and omohyoid muscles.

THE SYMPATHETIC NERVOUS SYSTEM

This system consists of—(1) A series of ganglia connected by intervening cords, extending from the skull to the coccyx. There are two sets of these ganglia, one on each side of the middle line of the body, partly in front of and partly on each side of the vertebral column. (2) Three great gangliated plexuses, situated in front of the spine, in the thoracic, abdominal, and pelvic regions. (3) Numerous smaller ganglia. (4) Innumer-

able nerve-fibers. (5) Numerous ganglia situated on, or con-

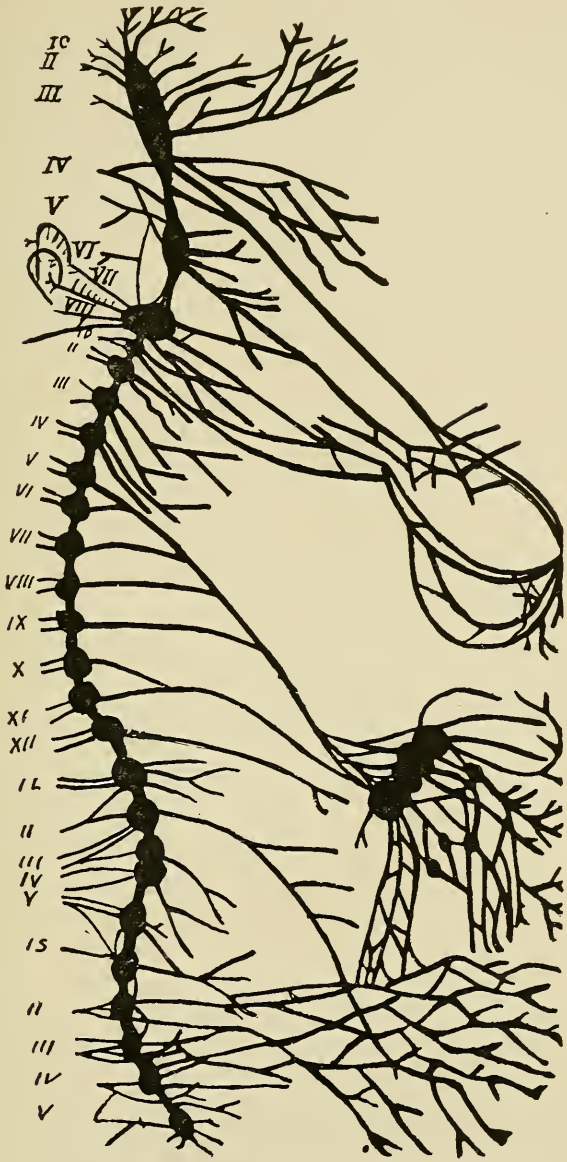


Fig. 77.—Diagram showing gangliated cord on one side, its connections with the spinal cord and with the various gangliated plexuses, etc.

nected with, the cranial nerves and their branches of distribution (Fig. 77).

Each gangliated cord may be traced upward from the base of the skull into the cranial cavity by ascending branches, which pass through the carotid canal, forming a plexus on the internal carotid artery and communicating with the Gasserian ganglion.

In the cervical region there are three sets of ganglia. These connect with the central nervous system through the first and second dorsal roots. In the dorsal region there are twelve pairs of ganglia; in the lumbar, four; and in the sacral, four or five. In the dorsal and lumbar regions the ganglia are connected with a corresponding segment of the spinal cord. In the pelvis the cords converge and form a single ganglion in front of the coccyx.

The three gangliated plexuses—the cardiac, in the thoracic cavity; the solar, in the upper abdominal cavity; and the hypogastric, in front of the promontory of the sacrum—consist of collections of nerves and cells. They distribute branches to the viscera of the thorax, abdominal cavity, and pelvis, and receive nerve-fibers from the gangliated cords and the cerebrospinal nerves.

Many smaller ganglia are situated in the various viscera—the heart, the lungs, the stomach, the uterus, etc. The numerous nerve-fibers that help to make up the sympathetic system are of two kinds—*communicating*, by means of which the ganglia communicate with each other and with the cerebrospinal nerves; *distributory*, by means of which sympathetic fibers are supplied to all the internal viscera, the muscular coats of the blood-vessels, the glandular structures, special organs, and unstriped muscles.

From the thoracic portion of the gangliated cords are given off the splanchnic nerves, which communicate with the solar plexus. The cardiac plexus is situated at the base of the heart. The epigastric or solar plexus is behind the stomach, in front of the aorta and the crura of the diaphragm. The hypogastric plexus, situated in front of the promontory of the sacrum, distributes branches to the viscera of the pelvic cavity.

When fully developed, the sympathetic system is found to consist of two divisions: The first division, or sympathetic system proper, often called the vertebral sympathetic, consists

of a chain of ganglia—the cervical, dorsal, and lumbar. These ganglia are connected with the spinal cord and give off fibers that pass peripherally, as a rule, through the medium of secondarily formed plexuses or ganglia, to the viscera of the thoracic and abdominal cavities, to the vascular system, to the skin with its glands and appendages, and to the pelvic viscera and genitalia.

The second division of the sympathetic is known as the parasympathetic or autonomic system. The fibers that make up this system come from the midbrain and enter the ciliary ganglion. A second set comes from the glossopharyngeal and the pneumogastric and their ganglia, and a third set from the lumbosacral part of the spinal cord.

The Functions of the Sympathetic Nervous System.—The sympathetic system appears to give a double nerve supply to the skin and its appendages, the glands, the vascular system, the alimentary system, and the respiratory system. It would appear further that these two divisions—the vertebral sympathetic and the autonomic—are mutually antagonistic in their functions. For example, impulses passing through the autonomic fibers by the oculomotor nerves of the ciliary ganglia to the sphincter of the eyes cause pupillary contraction. Impulses passing through the vertebral sympathetic, reaching the eyes through the carotid plexus and superior divisions of the fifth nerve, cause pupillary dilatation. The vertebral sympathetic contains the constrictors for the blood-vessels, whereas the vagus contains the dilators.

The vertebral sympathetic contains and transmits impulses that relax the smooth muscle-fibers of the esophagus, the stomach, etc. The pneumogastric transmits impulses that cause these organs to contract. In the pelvis the autonomic fibers transmit impulses that cause contraction of the smooth muscles of the colon, rectum, urethra, bladder, etc., whereas the vertebral sympathetic fibers transmit impulses that cause relaxation of these muscles.

It has been more or less definitely shown that the vertebral sympathetic is connected with the spinal cord by means of the white rami communicantes which leave the cord by the anterior roots immediately after the formation of the mixed nerve.

There is apparently every reason to believe that the sympathetic system is under the dominance of the higher centers. It is probably little, if at all, influenced by the will, but is certainly dominated by the moods and emotions. Anger, fear, pain, etc., manifest themselves outwardly by sympathetic disturbances, such as pallor, flushing, perspiration, dryness of the mucous membranes, pupillary, cardiac, and respiratory disturbances, and the like. The sympathetic system also possesses afferent fibers, which pass by way of the posterior roots to the spinal cord.

Under normal conditions the functional activity of the viscera is not reflected upon consciousness. In diseased states of the viscera sensory impulses originating in them are reflected upon consciousness as proceeding from areas of the skin supplied by the spinal nerves connected with the segments that receive the abnormal sympathetic impulses. This superficial skin localization, corresponding to the various organs, has been carefully worked out by Head and others. Furthermore, in certain diseased conditions, as, for example, in neurasthenia and in hysteria, in which the individual complains of peculiar sensations, pain, etc., in his viscera, it would seem that these afferent sympathetic impulses were reflected directly upon consciousness.

This would seem to indicate that the tone of the various organs of the body—of the circulatory system, the skin, the glands, the genito-urinary system, etc.—is maintained by the constant antagonistic action of the autonomic system, on the one hand, and the vertebral sympathetic system, upon the other. It is a disturbance of the balance of this antagonistic action, as the result of disease, exhaustion, toxemia, emotional distress, etc., that gives rise to the various symptom-complexes and diseases attributed to disturbed activity of the sympathetic nervous system. As an example may be mentioned the vasovagal attacks of Gowers: these are characterized by periodic seizures, which in some cases resemble epilepsy. They begin, as a rule, with a peculiar epigastric sensation, followed by palpitation and forcible heart action, and sometimes by a sensation as if the heart were ceasing to beat, a sense of suffocation, difficulty in breathing, a fear of impending death or as if some great calamity

were about to occur. Simultaneously there may exist coldness of the extremities, succeeded by a clammy perspiration or a burning and flushing all over the body. The attack may be accompanied by trembling and quivering of the entire body, and may last from a few minutes to several hours.

Further consideration of the sympathetic system shows an intimate relationship between the sympathetic and the secretions of certain of the ductless glands, which belong to the chromophil system. Chromophil cells are found in the medulla of the suprarenal glands, in the sympathetic ganglia and plexuses, in the kidneys, accessory suprarenals, carotid bodies, etc. The secretion of the chromophil tissue is capable of causing a characteristic reaction in structures which are innervated by the sympathetic system. For example: adrenalin, which is a product of the suprarenal medulla, acts solely on the tissues innervated by the vertebral sympathetic, causing a rise in blood-pressure, dilating the pupils, and exciting to increased activity the salivary and lacrimal glands, etc.

It is through the normal activity of the vertebral sympathetic system, the autonomic system, and the secretions of the chromophil glandular system that normal vascular tone, normal secretions, and normal activity of the cardiac, respiratory, digestive, and urinary systems are brought about and a general state of well-being and health is maintained. Disturbance of the normal activity of the sympathetic nervous system gives rise to a feeling of general discomfort, which manifests itself as nervousness, depression, irritability, anxiety, etc.

Disturbance in the functions of the sympathetic may also cause pupillary anomalies and disturbances, trophic and vasomotor disorders, which may evidence themselves in pathologic alterations in the tonus of the peripheral vessels, giving rise to such conditions as erythromelalgia, Raynaud's disease, migraine, intermittent claudication, angioneurotic edema, and the like.

EXAMINATION OF THE PATIENT

HISTORY AND PHYSICAL EXAMINATION

It is especially important, in the study of patients who are suffering with nervous and mental disorders, that a thorough, complete, and correct history be obtained. This is at times difficult to obtain and consumes much time. In some cases the physical and clinical examination of the patient is sufficient to establish the diagnosis, whereas in other cases the diagnosis can be made only from a study of the history.

The following is an outline for obtaining the history and for making an examination of a patient:

The name. Age. Sex. Civil state. Date.
Address. Occupation.

Complaint: The patient should be allowed to state in his or her own words, without suggestion on the part of the examiner, the abnormality of function for which he or she has consulted the physician. In cases in which the patient is unable to talk, or the age or mental condition precludes the possibility of securing a proper statement, the nature of the complaint should be ascertained from relatives and friends.

It has been my experience that very frequently the patient's statements regarding the complaint are not accurate, and one must depend upon careful inquiry later on in the history for correct and enlightening information.

Duration: Care must be exercised in accepting the statement as to the duration of the illness.

Number of attacks: If the first, second, third, etc.

Progress: Is the condition getting better or worse?

Course: Is the patient improving, or is he or she growing rapidly or slowly worse, or is the course stationary.

Family History: Under this head careful inquiry should be made as to the existence of nervous or mental diseases, tuberculosis, rheumatism, heart or kidney affections, venereal disease, drug addictions, alcoholism, etc., existing in the grandparents, the parents, the brothers and sisters, uncles, aunts, or other members of the patient's family. Inquiry should also be made as to the age at which members of the immediate family died, and as to the cause of death in each case. The object in making a careful inquiry into the existence of disease in the family or the ancestors is to learn whether the affliction is one of a certain number of nervous disorders known as familial diseases, and which occur usually in succeeding generations. There are also numbers of diseases that show a marked hereditary tendency, existing in the parents and children. Chronic disease, drug addictions, alcoholism, unfavorable living conditions, etc., in the parents predispose to the occurrence of nervous and mental affections in the offspring.

Past History: Careful inquiry should be made into the past history to ascertain the living conditions, the health, the occurrence of injury, worry, shock, etc., experienced by the mother during the time she was pregnant with the patient. The conditions under which the patient was born, whether the labor was prolonged, difficult, instrumental, or normal. The diseases which occurred during infancy and childhood, whether they were attended by any unusual symptoms, or whether they were followed by complications, and if so, how these complications affected the individual, whether the individual took his nourishment normally, the age at which the teeth first appeared, when he first began to crawl, walk, and talk; the occurrence of convulsions, of injury, or of skin eruptions; the progress made at school, as to physical development. During later childhood, was there any disease, injury, convulsions, or venereal infection? if a female, the age at which menstruation first appeared, its character, and the effect its occurrence had upon the general disposition of the patient. The age at which the appearance of the patient and the development of the sexual organs indicated a change from childhood to adolescence. Inquiry should be made as to the occurrence of disease later in life,

of injury, of the habits as regards alcohol, drugs, work, and recreation, and the existence of venereal disease. The number of children, the number of miscarriages and the causes that brought them about, the age at which the patient was married, the state of health of the husband or wife, of the children, the home conditions, whether congenial and happy or irritating and unhappy. It is important, in eliciting the past history of the individual, to assist, by judicious questioning, the memory of the patient or his friends, so that an accurate account of the past life of the patient may be obtained.

It is important to remember that the patient may unintentionally give unreliable information. If, therefore, the history is one of unusual conditions, it should be confirmed by further questioning as to details.

Present Illness: It is most important, in getting an account of the illness for which the patient consults a physician, to secure this information in as nearly a correct chronologic order as possible. Although it is permissible, and even advisable, to allow the patient to state his complaint in his own words, it is necessary at this time to insist upon his giving you an accurate account of his symptoms.

I find it advisable to have the patient or his friends state the first indication that made him think he was ill, the mode of onset, whether it began gradually or developed suddenly, whether intermittent or paroxysmal in character, if there were intervals between the symptoms, the length of the intervals, etc. I then have the patient recount, as well as he is able, when and how subsequent symptoms manifested themselves, also the distribution of the symptoms,—whether one part, several parts, or the entire body was affected,—and the progress of the illness, from the date of onset up to the present time.

Having obtained a correct and full history from either the patient or his friends, preferably from both, the next step is to make a thorough examination of the patient. I wish to emphasize here the importance of making a careful and complete physical examination, for it is my belief that if proper physical examinations were made oftener, fewer cases would be diagnosed as neurasthenia, hysteria, functional neurosis, etc., and many

would be found to be diseased conditions of the kidneys, heart, blood-vessels, blood, ductless glands, etc.

PHYSICAL EXAMINATION

The following points should be noted: The appearance, the expression, and the weight of the patient. The blood-pressure should be taken and the intelligence tested. Some idea of the intelligence of the patient is obtained in taking the history—whether or not the patient comprehends well and quickly; whether his thinking processes are normal or slow, and whether the comprehension is poor and the individual stupid. In arriving at a correct conclusion regarding the intelligence of the patient, one must always take into consideration the age, the station in life, etc.

The Skin.—This should be examined for scars, eruptions, or swellings. The state of its nutrition—whether it is thin and glossy, or thick and scaly; whether moist or dry; the presence of pigmentation or if absorption of pigment has taken place; the state and condition of the skin appendages—the hair and nails.

The Bones and Joints.—Note the presence of any deformities, fractures, diseases of the joints, or malformations.

The Mouth, Nose, and Throat.—The condition of the teeth and of the tonsils should be learned, as well as the presence of eruptions, ulcerations, or scars on the mucous membranes of the mouth, tongue, nose, or throat; the presence of adenoids; the amount of breathing space; abnormalities in the nose and palate.

The Eyes and Ears.—Are the eyes set normally in the orbits, or is there a protrusion or sinking, swelling, etc.? Ascertain whether the ocular movements are normal, and whether the pupils are regular, irregular, and active. Determine whether the ears are properly set, if there are any deformities or tophi, and whether the two ears are symmetric in shape.

The Respiratory System.—Careful examination should be made of the lungs and of the larynx.

The Cardiovascular System.—A thorough examination should be made of the heart, giving attention to its size and position,

the presence or absence of murmurs, the rate and the rhythm of the heart-beat. The blood-vessels should be examined for the presence of sclerosis, degeneration, etc. In examining the blood-vessels it is important to learn the condition of the temporals, the radials, the dorsalis pedis arteries, the aorta, and the veins.

Glandular Enlargements and Tumors.—The entire body should be carefully examined for the presence of glandular enlargements or tumors.

Gastro-intestinal Symptoms.—Careful examination of the functions and activities of the stomach and intestines should be made in those cases in which the symptoms point to the gastro-intestinal tract. The absence or presence of constipation should also be noted.

Genito-urinary System.—In all cases complete examination, both macrosopic and microscopic, of the urine should be made, including the test for indican. Where indicated, a careful examination of the sexual organs, the urethra, the bladder, the ureter, and the kidneys should be made.

The Rectum and Pelvis.—Careful inquiry should be made as to the presence or absence of hemorrhoids, fissures, ulcerations, and, in women who have borne children, as to the condition of the perineum, etc. When symptoms point in this direction, an internal examination is to be made. Purulent discharges from any portion of the body should be carefully examined microscopically.

The Blood.—The examination of the blood should include a differential count, the hemoglobin estimate, and a white and red cell count. The Wassermann test is often important and many times is absolutely necessary.

The Cerebrospinal Fluid.—This should be examined in all cases that are difficult of diagnosis or where there is any question as to the cause of the illness, and in cases in which this examination is indicated. This examination should include a cell count, the sugar test, the Wassermann test, the specific gravity, etc.

In many cases it is well, before proceeding to make an examination of the nervous system, to make a test of the mentality of the patient.

The Memory.—By proper questioning one can readily determine whether the memory is disturbed and if the disturbance is diffuse or is impaired only for recent or remote events.

Orientation.—By proper questioning it is simple to determine if the patient recognizes his surroundings, *i. e.*, orientated for place; whether he recognizes persons around him, *i. e.*, orientated for persons; and whether he is familiar with the passage of time, such as time of day, day of week, etc., *i. e.*, orientated for time.

Illusions, hallucinations, delusions can be discovered by questioning the patient, or by careful inquiry from the relatives and friends.

Disturbances of Conduct and of the Emotions.—These may be discovered during the course of the examination, or a history of the same may be obtained from the family or friends.

EXAMINATION OF THE NERVOUS SYSTEM

Speech.—In examining for speech defects one should note any disturbance of phonation, the existence of stuttering, explosive or monotonous speech. The tone of the voice, whether low or loud, and the elision of syllables or words should be noted; also whether any of the various types of aphasia exists. In examining for speech defects one must ascertain whether the patient can or cannot voluntarily express ideas in words; whether he can name objects that he sees; whether he can express written sentences in words or repeat words or sentences that he hears. Voluntary speech is disturbed in stuttering, stammering, in some diseased conditions, such as weakness or paralysis of the larynx, vocal cords, tongue, lips, and pharynx, in general bodily weakness, etc.

When the individual is unable to repeat test words, as, for example, "statistician," "perturbation," "German riding artillery brigade," etc., without the elision or reduplication of syllables, letters, or words, there exists a condition in which the higher cerebral associative centers are involved. When the individual is unable to express his ideas in words, whether these ideas originate in the visual field, in the sensory field, on in the

intellectual field, and there exists no physical impediment, such as local diseases of the tongue or larynx, etc., for the expression of these ideas, a condition known as aphasia exists.

Gait.—The individual should be asked to walk, and any abnormality noted. The gait may be uncertain, ataxic, staggering, hemiplegic, spastic, steppage, shuffling, diplegic, etc.

Examination of the Motor System.—*Muscular Development and Muscular Nutrition.*—Careful examination should be made for the presence of muscular atrophy or hypertrophy, and if either is present, the degree and the location should be carefully noted. The limbs and the two sides of the body should be compared with each other by actual measurement.

Motor Power.—The strength of the flexors and extensors of the extremities and of the two sides of the body should be compared, and any weakness or paralysis should be carefully noted as to extent and distribution.

Muscle Tone.—Examination should be made to ascertain whether the muscle tone is increased, decreased, or normal, and if disturbed, the degree and distribution should be noted. Abnormalities are discovered by comparing the two sides of the body with each other, if only one side is diseased, or if there is a general involvement, by comparing the state of the muscle tone in the affected individual with the muscle tone of a normal person.

Abnormal Movements.—These may occur constantly or at intervals. They may be very limited in extent, being insufficient to bring about twitching of the trunk or extremities, or they may be so extensive as to occasion violent contractions of the trunk or extremities.

The following are the types of abnormal movements: *Fibrillary tremors*, which are contractions of muscle-fibers or groups of muscle-fibers, producing wave-like twitchings in the muscles. They occur in degenerative diseases of the spinal cord, in which there is a gradual involvement of the anterior horn cells; also in toxic conditions and upon exposure to cold, etc.

Other Forms of Tremors.—Tremors may be rapid or slow in rate, and they may be limited or wide-spread in extent. They may be *passive;* that is, occur while the affected part is at rest, or

they may be of the *intention* type, that is, occur during the voluntary movements of the affected part. Some passive tremors can be inhibited upon voluntary movement, and some are rendered very much worse during voluntary activity. Nearly all tremors are intensified upon emotional excitement. Tremors occur very commonly in both general and nervous diseases. They are a prominent symptom in senility, exophthalmic goiter, paralysis agitans, multiple sclerosis, general paresis, etc. They may affect any portion of the body, but most frequently involve the upper extremities, the tongue, the lips, and the head.

Choreiform Movements.—These movements are irregular, purposeless, inimitable muscle contractions, which occur at irregular intervals, varying in extent from movements so mild as to be perceptible only under careful observation, to movements that are so exaggerated as to result in violent contractions of the entire body. These movements usually involve the upper extremities and the face, but may involve any portion of the body.

Athetoid Movements.—These are caused by involuntary muscle contractions, which bring about wave-like or worm-like movements of the distal parts of the extremities and sometimes of an entire extremity. These movements are not under voluntary control, and are due to cerebral disease, occurring most frequently in congenital or early acquired cerebral disorders (Figs. 78, 79).

Spasms are contractions of muscles or groups of muscles due to either a direct or a reflex stimulation of a nerve. They are usually limited in extent.

Convulsions are more or less extensive contractions and relaxations of groups of muscles, which bring about movements of the extremities, head, trunk, or entire body. They may or may not be attended by loss of consciousness, and are due to either direct or reflex irritation of the cerebrospinal motor centers.

Coördination is tested for by having the patient touch the tip of his nose with the finger with his eyes closed; by having him bring the two index-fingers together, making the arms describe the arc of a large circle, with his eyes open and then with his eyes closed; by having him touch the knee of one side with the heel of the foot of the opposite side, directing him to ele-

vate the leg and bring the heel directly down so as to touch the knee; this should be done with the eyes both open and closed.

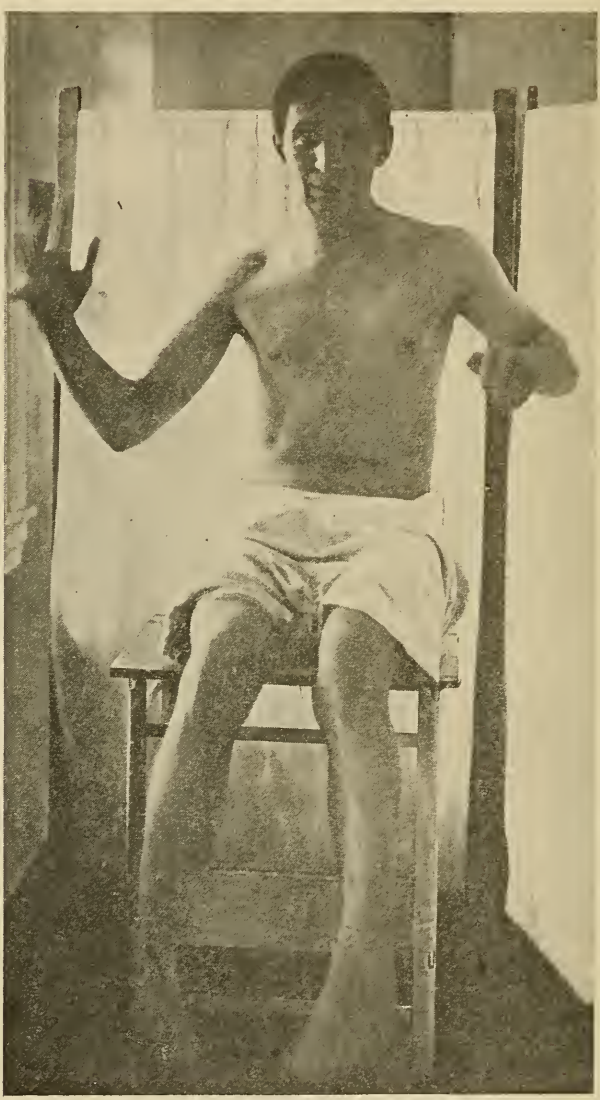


Fig. 78.—Patient showing athetoid movements and facial spasm.

Romberg's phenomenon is a test for coördination and equilibrium. The patient should stand with the feet close together, both at the heels and toes, and with the eyes closed.

In all these tests the inability of the patient to perform the desired action quickly and accurately is an indication that there is an interference with the reception of sensory stimuli, the transmission of these stimuli to the higher centers, the elaboration in the higher centers, or the ability of these stimuli to influence reflexly the motor mechanism controlling the desired action.

Diadokokinesis.—This is tested for by having the patient extend both hands in front of him, and rapidly rotate the hands

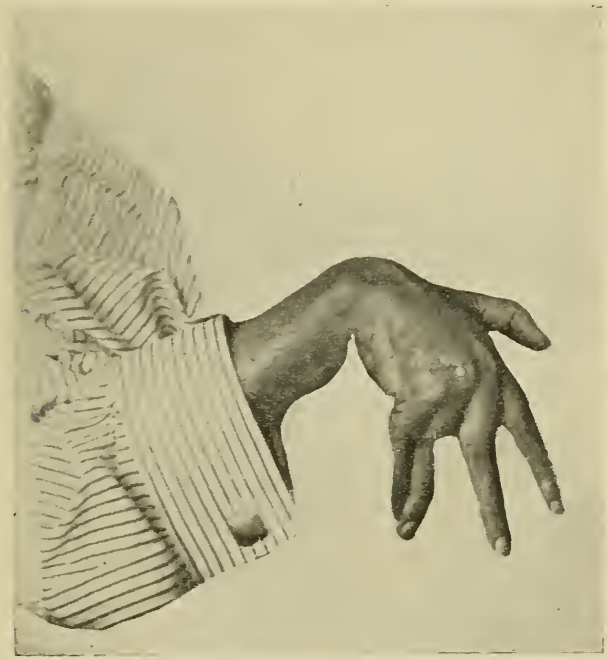


Fig. 79.—Hand of patient showing athetoid movements.

and forearms in their long axes. Under normal conditions this rotation is carried out regularly and equally in both arms. In diseased conditions in which there is marked interference with the cerebellar control of coördinate movements there is a disturbance in the regularity and similarity of rotation in the two arms. This condition is known as adiadokokinesis, and is evidenced by an irregular and unequal rotation of the two arms.

REFLEXES

The Deep Reflexes—The important deep reflexes are those of the knee, the tendo Achillis reflex, the triceps, the biceps, the periosteoradial, the jaw reflex, and the corrugator supercilii reflex. These reflexes are tested for by having the muscles connected with the part of the body to be tested in a state of partial tension. The individual's attention should be distracted from the part of the body under examination, and a

Fig. 80.—Position of patient in testing for tendo Achillis reflex.

quick sharp tap should be made upon the tendon connected with the muscle to be tested.

The Tendo Achillis Reflexes.—The patient may kneel on a chair, with both feet extending over the edge. The tendo Achillis should be tapped with some instrument, preferably a pleximeter. When the reflex is present there will be a sharp, quick contraction of the posterior leg muscles and the foot will

be extended If the patient is in bed this reflex can be tested for by slightly flexing the leg on the thigh, grasping the foot, and making slight traction on the posterior leg muscles by flexing the foot on the leg, distracting the patient's attention, and tapping the tendo Achillis (Figs. 80, 81).

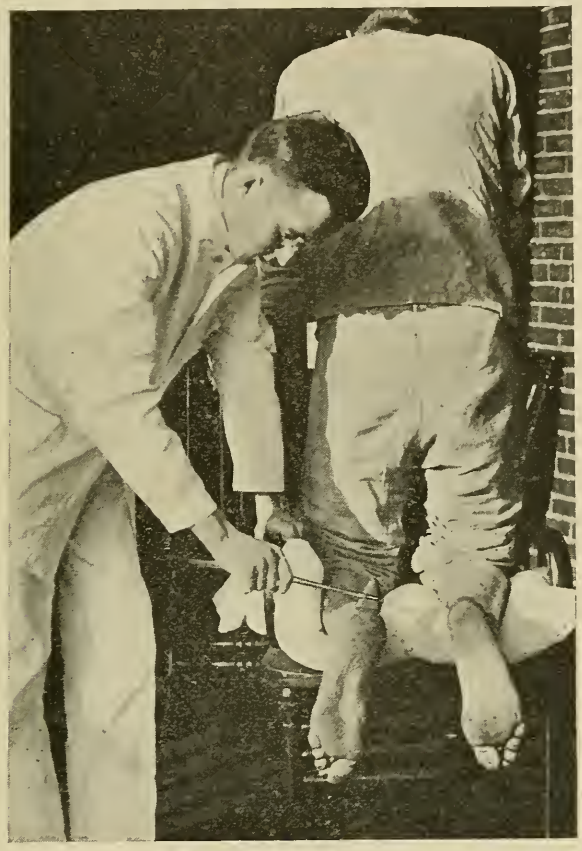


Fig. 81.—Position of patient in testing for tendo Achillis reflex.

The Knee Reflexes.—The patient should preferably be in the sitting position, with the leg flexed on the thigh, at an angle of about 100 degrees, or he may cross the legs and allow one leg to swing loosely over the other (Figs. 82, 83). On tapping the tendon of the quadriceps extensor muscle, if the reflex is present, a contraction results, bringing about a more or less marked extension of the leg.

The Periosteoradial Reflex.—This is tested for by having the patient pronate the hand on the forearm and flex the forearm on the arm. The examiner may either support the forearm in his hand or, better still, have the patient sit in an arm-chair and allow the arm to rest on the arm of the chair (Fig. 84). By tapping sharply on the radius, just above the wrist-joint, if

Fig. 82.—Position of patient in testing for knee reflexes.

this reflex is present, there results an extension of the hand on the forearm and a contraction of the supinator longus muscle.

The Biceps Reflex.—This reflex is best tested for by having the forearm semiflexed on the arm. The examiner should then place his finger over the biceps tendon (Fig. 85), and tap the finger over the tendon with a pleximeter; when this reflex is present, there results a contraction of the biceps and sometimes flexion of the forearm.

The Triceps Reflex.—The presence or absence of this reflex is tested for by having the patient flex the forearm on the arm (Fig. 86). The examiner may then either support the arm or the arm may rest over the back of a chair. The tendon of the triceps muscles, immediately above its attachment to the olecranon, is tapped with a pleximeter; when this reflex is present, there occurs a contraction of the triceps muscles and an extension of the forearm.

Fig. 83.—Position of patient in testing for knee reflexes.

The *jaw reflex* is best tested for by having the patient relax the muscles of mastication slightly, thereby allowing the jaws to be slightly separated. The examiner then places his finger upon the lower jaw, between the lip and the chin (Fig. 87). The patient's attention is distracted, and a sharp, quick tap is made upon the examiner's finger; when the jaw reflex is present, there is a sudden closing of the jaws.

The *corrugator supercilii reflex* is best tested for by having the patient look downward and relax the upper eyelids. A gentle but quick tap is given just above the eyebrows, about $\frac{1}{4}$ to $\frac{1}{2}$ inch from the midline; when this reflex is present, following the tap there occurs an elevation of the eyebrows.

The deep reflexes may be present and yet be difficult to bring out. This is often due to the fact that it is impossible for the

Fig. 84.—Position of patient in testing for periosteoradial reflex.

patient to distract his attention from the part under examination, and he consequently voluntarily inhibits the production of the reflex. Under these conditions the *Jendrassik* method of reinforcement may be successful. This consists of distracting the patient's attention to another part of his body by having him voluntarily exert some motor activity not connected with the part under examination; thus, in testing the lower extremities, he is requested to clasp his two hands together, and at a

word of command from the examiner to exercise as much pressure as possible, with his hands against each other (Fig. 88). His voluntary efforts are thus directed toward the movements of his arms and hands while his legs are under examination. In examining the upper extremities, the attention my be directed toward movements of the legs.

The deep reflexes are increased in all conditions in which the inhibiting power from the higher centers is lessened or destroyed,

Fig. 85.—Position of patient in testing for biceps reflex.

except in complete transverse lesions of the spinal cord, The deep reflexes are decreased or lost in all conditions in which there is an interference with or a destruction of the reflex arc.

The Superficial Reflexes.—The important superficial reflexes are the plantar, the Babinski, the cremasteric, the abdominal, the corneal, and the pharyngeal.

The *plantar reflex* is tested for by irritating the sole of the

foot with the examiner's fingers, or lightly with some object. As a result of this stimulation there occurs first a flexion of the toes. In sensitive individuals, or where the stimulation has been excessive, in addition to the flexion of the toes there is a flexion of the foot, and in some cases a retraction of the entire leg (*retraction reflex*).

The *Babinski reflex* is tested for by stroking the outer side of the plantar surface of the foot firmly from the heel toward the

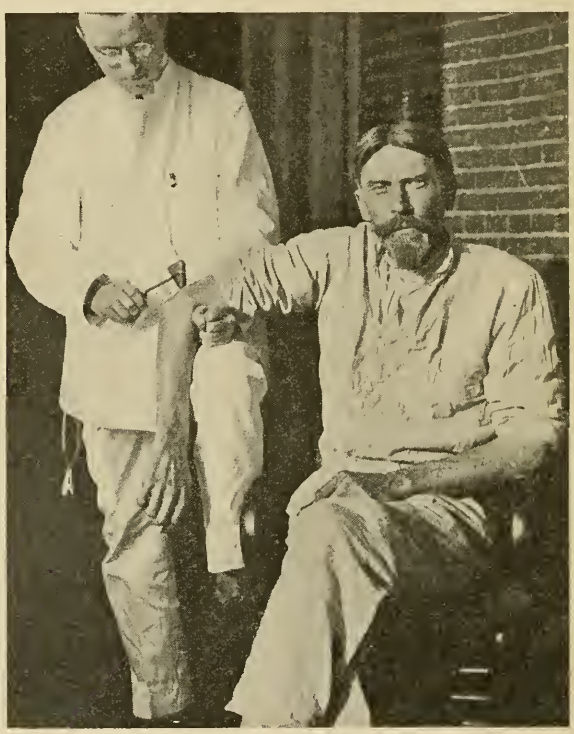


Fig. 86.—Position of patient in testing for triceps reflex.

toes (Fig. 89); when this reflex is present, there occurs a dorsal extension of the great toe, and in perfectly typical cases a fan-like spreading out of the other toes. Oppenheim and Gordon have each described a method for producing the same reflex—the former, by irritating the inner side of the lower end of the tibia, and the latter, by irritation and pressure along the outer surface of the tibia. This reflex is present normally in children less

than two years old and sometimes even in older children. When this reflex is present, except in the case of infants, it is practically always indicative of a disease process that has resulted in destruction of or caused an interference with conduction in the corticospinal motor pathways.

Fig. 87.—Position of patient in testing for jaw reflex.

The *cremasteric reflexes* are best tested for by gently stroking the inner aspect of the upper thigh, which causes retraction of the testicle upon the same side.

The *abdominal reflexes* are tested for by quickly and lightly stroking the abdominal walls. This brings about a contraction of the abdominal muscle situated beneath the irritated area, and we thus may have the hypogastric or epigastric abdominal reflex.

The *corneal* and *conjunctival reflexes* are tested for by suddenly approaching an object toward the eyes, or by lightly touching the conjunctiva or cornea, which produces a quick contraction of the sphincter palpebra.

The *pharyngeal reflex* is best tested for by touching the base of the tongue or the pharyngeal walls; when this reflex is present,

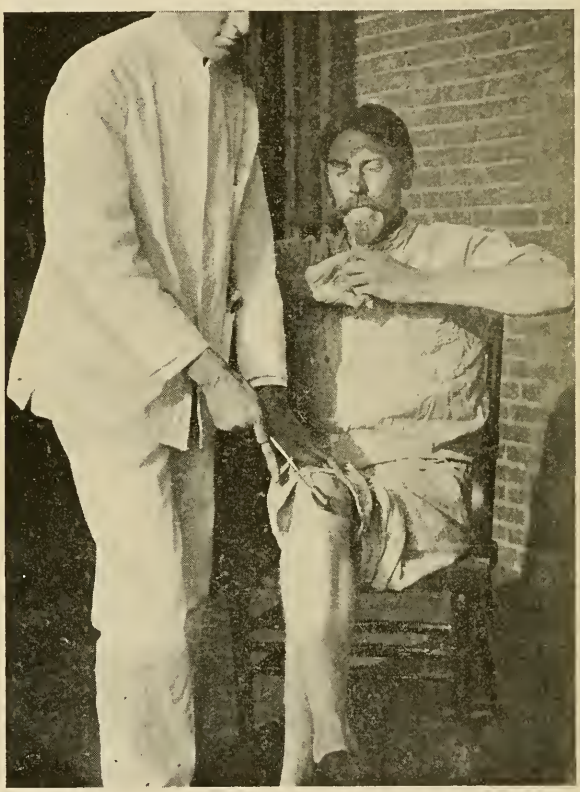


Fig. 88.—Illustrating Jendrassik's method of reinforcing the reflexes.

there occurs a contraction of the pharyngeal muscles, retching, and spasm of respiration.

The superficial reflexes may be increased in the absence of any pathologic conditions, and, in fact, they are frequently increased in normal individuals. The absence of the superficial reflexes may occur in organic diseases; in which there is a loss of sensation, where extensive destructive processes have taken

place in the corticospinal pathways, in functional neurosis, and in various diseases other than those of the nervous system. The loss of the superficial reflexes, with the exaggeration of the deep reflexes, is always very suggestive of an organic disease of the central nervous system.

Organic Reflexes.—*Rectal Reflex.*—The reflex activity of the rectal sphincter is best tested for by inserting the finger into the

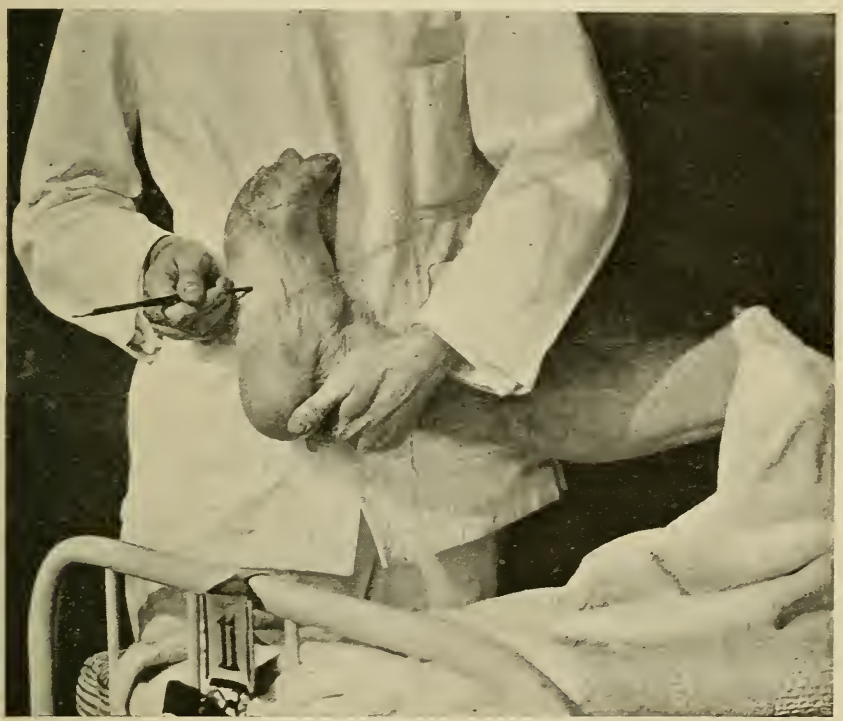


Fig. 89.—Babinski's reflex.

rectum. In those cases in which there is an increase in the rectal reflex the attempt will bring about a quick contraction of the sphincter; where the rectal reflex is lost, the examining finger discovers a relaxation of the sphincter, and the presence of the finger does not produce contraction.

Vesical Reflexes.—In exaggeration of this reflex there occurs retention of urine, with dilatation of the bladder. Where the reflex spasm of the sphincter muscle is not too extreme, after

moderate dilatation there occurs a constant dribbling or an occasional involuntary emptying of the bladder. Where the reflex spasm of the sphincter is excessive, rupture of the bladder wall may take place unless the bladder be emptied by catheterization. Where the vesical reflex is lost, relaxation of the vesical sphincter and constant dribbling of urine occur.

Where voluntary control of the rectal and vesical sphincters is lost, if attended by spastic contraction of the sphincters, this is due to a pathologic process that affects the corticospinal pathways; when the loss of control is accompanied by a relaxed condition of the sphincters, the pathologic process involves the reflex arc between the bladder or rectum, or both, and the lumbar enlargement of the spinal cord.

SENSORY FUNCTIONS

Epicritic or Light Sensibility.—Under this form of sensation are grouped tactile sense, pain sense, temperature sense, and the ability to locate milder forms of stimulation.

Tactile Sense.—In testing for the sense of touch one uses a camel's-hair brush, a feather, a bit of absorbent cotton, or any object with which a light touch may be made. The patient is directed to close the eyes, and to indicate promptly, either by word or action, that he is conscious of the touch. The rapidity of perception, the ability to locate the stimulus, and a comparison of the correctness of the perceptions in similarly situated areas are to be noted.

Pain Sensation.—Similar tests are to be made for the perception of pain sensation, using in this case a sharp and a blunt object, such as the point and head of a pin. Rapidity of perception, the location of the stimulation, and the comparison of the degree of stimulation in similarly situated areas are to be noted.

Temperature Sensation.—This form of sensation is to be tested for by using two test-tubes, the one filled with hot and the other with cold water. The temperature of the hot water should be between 105° and 110° F.; that of the cold water should be about 70° F. If the temperatures are very much above or below

these points, the patient may perceive them as pain, rather than as heat and cold. The various portions of the body should be touched alternately with the tube containing the hot and the cold water, and the patient is to indicate which one is being used. In making this examination the rapidity of the perception, the correctness, the ability to localize, and the comparison in similarly situated areas in the two sides of the body are to be noted.

Protopathic or *coarse sensibility* is that form of sensibility whereby the patient is conscious of stimulations that are much more severe than those perceived in epicritic sensibility. This form of sensibility is often retained when epicritic sensibility is lost, and in the return of sensation after disease the protopathic sensibility reappears before the epicritic sensibility. It is tested for in the same way as epicritic sensibility, with the exception that the stimuli must be more severe; thus in testing tactile sense, instead of barely touching the surface with a camel's-hair brush, etc., the application must be made with more force. In testing pain sense the stimulations are applied more severely, and in testing temperature sense the test-tubes should contain very hot and very cold water.

Deep Sensibility.—Muscle Sense.—This should be tested for in both upper and lower extremities. The patient closes his eyes, and the examiner lightly grasps a toe and flexes or extends the same, while the patient indicates the direction in which the movements are being made. The same tests are to be made with the ankle- and knee-joints, the fingers, the wrist, the elbow, etc.

Paresthesias or Subjective Sensations.—Of these, numbness, tingling, formication, a sense of swelling or of smallness, burning, heat or cold, etc., may be complained of. The distribution and duration of these sensations should always be noted.

Tenderness of the nerve-trunks on pressure is to be examined for by compressing the superficial nerves between the examiner's fingers and adjacent bony structures—the ulna, the back of the internal condyle of the humerus, the anterior tibial, the back of the head of the fibula, the musculospiral nerve in its groove along the outer side of the humerus, etc., are among the nerves most easily tested for.

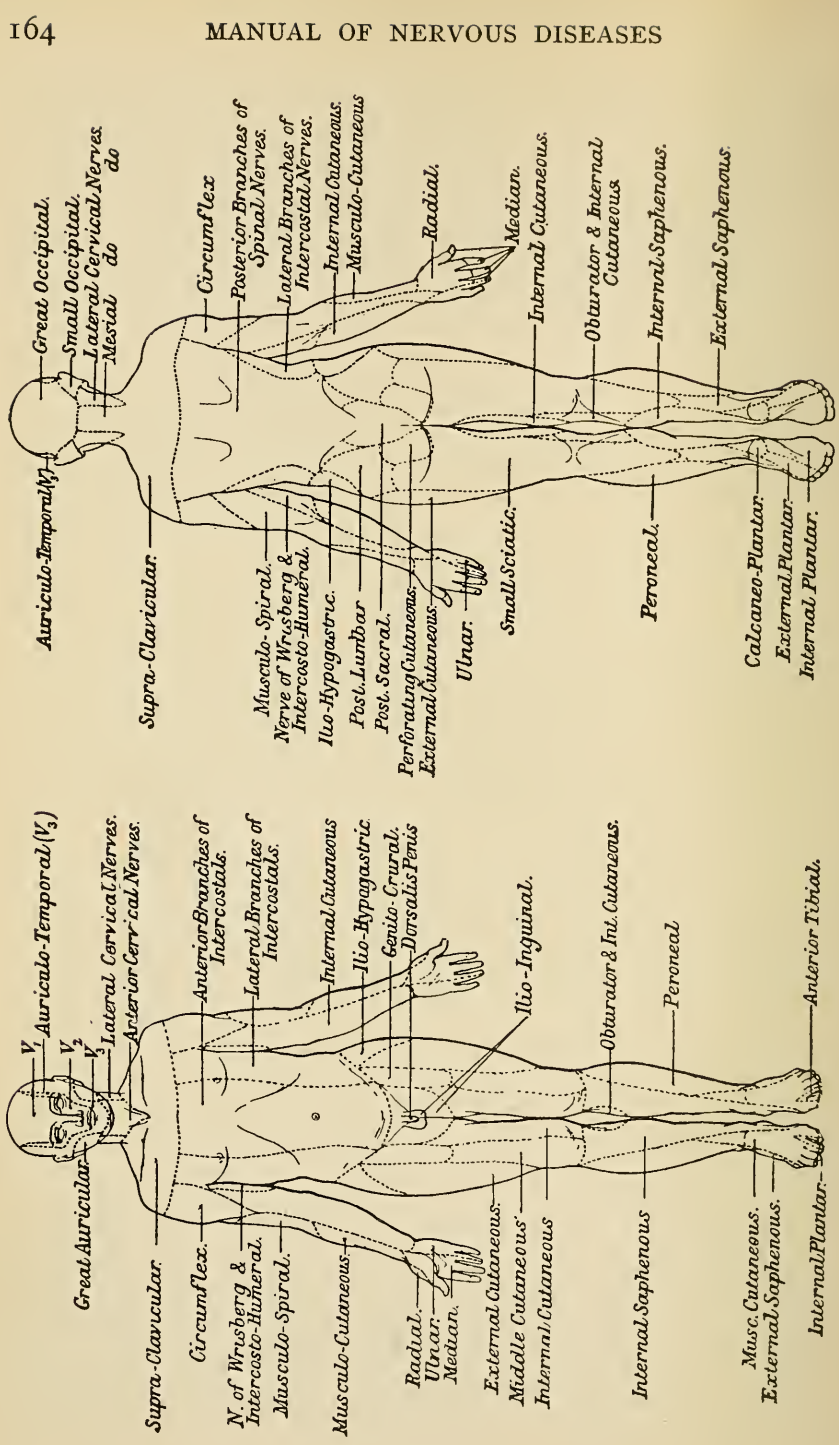


Fig. 90.—Diagram of cutaneous areas of the peripheral nerves (Fowler).

The Hysterogenic Zones.—Pressure should be made over the ovarian region, inframammary region, beneath the clavicles, over the apex of the heart, the epigastric region, etc. An abnormal sensitiveness to even light pressure in these regions is frequently present in functional conditions, but also occurs in diseased states of the underlying organs.

Stereognostic Sense.—This form of sensation is tested for by having the patient close his eyes, and then placing different objects in his hand. Under normal conditions the individual is able to name familiar objects from the various sensations imparted to him from the feel of these objects. An inability to recognize these objects is called astereognosis.

Apraxia.—This is a condition in which an individual, even if he is familiar with the name of an object and can describe its use, is unable to go through the movements that should accompany its use. This condition is always serious, as it indicates an extensive disintegration of the associative cerebral tracts.

Disturbance of sensation may occur as the result of organic or functional disease: when it is the result of functional disease, the distribution is such as would correspond to cerebral involvement; when it is due to organic disease, it may be caused by either irritation or destruction of peripheral nerves, spinal cord, brain-stem, or brain.

It is only from a study of the distribution, the character of sensory disturbances, and often after a careful consideration of other symptoms, that one is enabled to say definitely which portion of the central or peripheral nervous system is involved (Figs. 90, 91).

Vasomotor and Secretory Disturbances.—Examination is to be made to discover if abnormalities of the vasomotor balances are present: these are evidenced by cyanosis, abnormal paleness, abnormal redness, rapid transition from paleness to redness or vice versa, peculiar purplish appearance of the skin, delayed return of the normal color to the skin after pressure or irritation, and dermatographia.

Secretory disturbances evidence themselves in abnormalities of the secretions. They may be either increased or decreased, and this increase or decrease may be localized or general. In

rare instances there is a perversion of the secretions—instead of the normal color and odor the secretions become abnormal in color and odor.

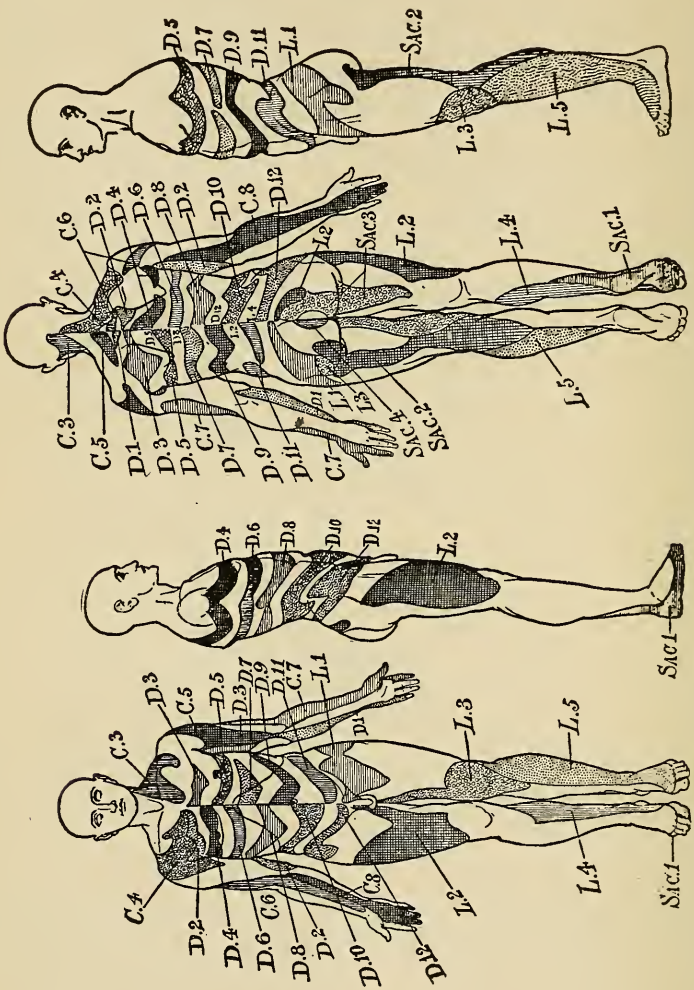


Fig. 91.—Sensory distribution corresponding to the various segments of the spinal cord (Head).

Trophic Disturbances.—These should be carefully examined for. They may affect the superficial tissues, giving rise to alterations in the appearance of the skin, falling out of the hair, change in the color of the hair, falling out of the teeth, a changed appearance of the nails, which latter may become brittle, ridged, thickened, etc. Ulcerations may affect the skin and the super-

ficial tissues, or the trophic disturbances may involve the deeper structures, affecting the bones, joints, muscles, etc., and giving rise to such conditions as perforating ulcers, Charcot joints, abnormal brittleness of the bones, etc.

ELECTRIC EXAMINATION

In making an examination of the muscles and nerves as to their electric excitability, one must have a faradic battery, with which there should be connected a resistance coil for increasing or decreasing the strength of the current; a galvanic battery, which will supply at least a current up to 30 milliamperes; a galvanometer; an automatic interrupter; two conducting wires; a number of electrodes, varying in size from 24 square inches to a small rounded electrode about $\frac{1}{2}$ inch in diameter, and a metal brush.

In order to make an electric examination it is necessary to be familiar with the areas at which the nerves and muscles respond most readily to electric stimulation; these areas correspond to the positions at which the motor or mixed nerves lie near the surface, or the points at which the nerves enter the muscles. The accompanying diagrams (Fig. 92) show the position of these points.

The nerves that are most readily accessible to electric stimulation are the facial, the median, the musculospiral, the ulnar, the anterior crural, the sciatic, and the anterior tibial nerves.

In making the examination one must be certain that the apparatus is in good working order. The electrodes are to be moistened with warm salt solution, and the patient's skin should be moistened with the same solution. The parts to be examined are to be placed in such a position that they can be observed well and are easily accessible. Furthermore, they should be in such position as to bring about relaxation of the muscles to be examined.

In making an examination one should test the affected muscles with the faradic and galvanic currents, in order to determine if there is increased or decreased response to either the galvanic

or the faradic current; whether there is a qualitative disturbance of the electric reaction, bringing about a partial or complete

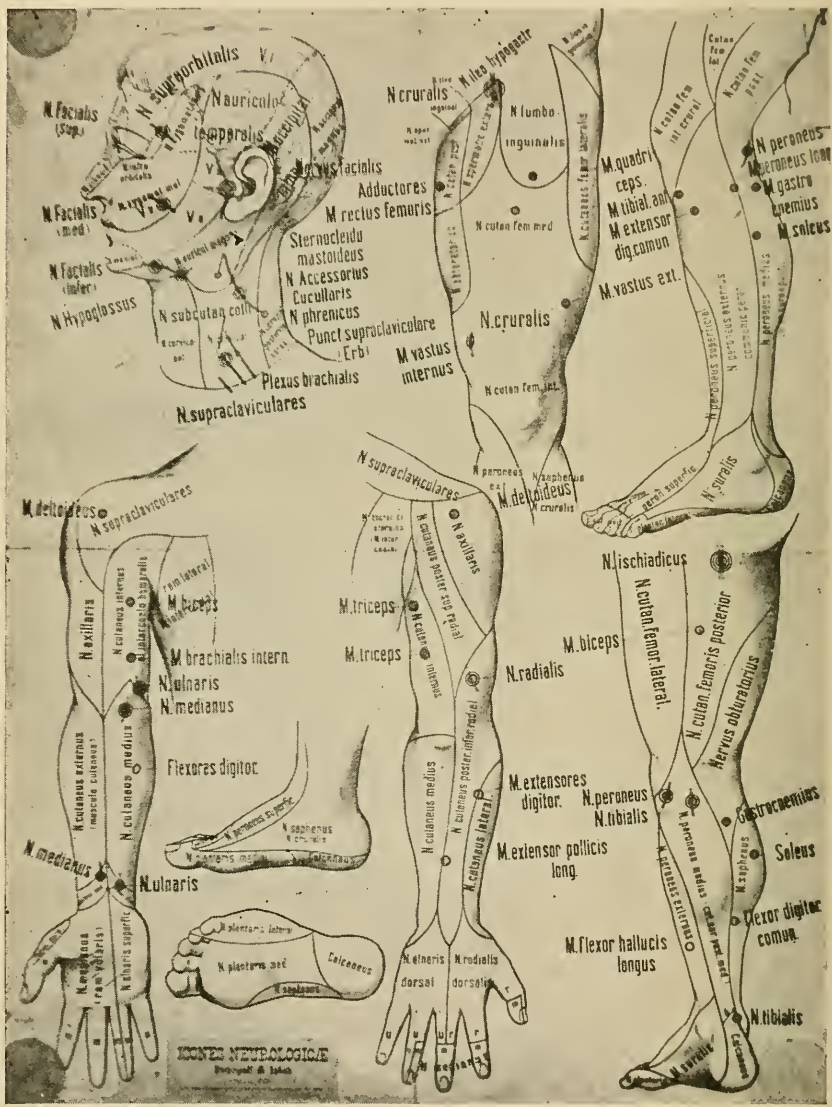


Fig. 92.—Areas at which the nerves and muscles respond most readily to electric stimulation.

reaction of degeneration. In special cases the myasthenic, the myotonic, and the tetanic reactions should be tested.

The faradic current is transmitted only by nerve-fibers; the galvanic is transmitted by both nerve-fibers and muscle tissue. It is the inability of the faradic current to be transmitted through muscle tissue in the absence of nerve-fibers that is responsible for the qualitative changes to electric stimulation in diseased conditions.

In making an examination with the faradic current one electrode—usually a large one—is placed at some indifferent point over the back, the sternum, etc.; the other electrode, which may vary in size from the smallest to one about $1\frac{1}{2}$ inches in diameter, is applied over the nerve or the excitable area of the muscle that is to be tested. The strength of the current is gradually increased until a muscle contraction occurs or until the examiner is satisfied that muscle contraction with this current cannot be induced.

Under normal conditions currents of very moderate strength produce a muscle contraction that is continuous until the current is interrupted, either upon stimulation of the nerve or of the muscles, the response requiring slightly weaker current upon stimulation of the nerve than upon that of the muscle.

In examining with the galvanic current one may use a large electrode, placed at some indifferent point, and a smaller electrode, about 1 inch in diameter, which is to be placed over either the nerve or the muscle that is to be tested. In some cases it is necessary to use two small electrodes, placing one over each end of the muscle under examination.

Under normal conditions, when the galvanic current is passed continuously through a nerve or muscle, no muscle contraction results, and it is only upon opening or closing the electric circuit that the contraction occurs. The contraction that results upon closing the circuit under these conditions is stronger than the contraction that results upon the opening of the circuit, and the contraction occurring at the negative pole is more marked than the contraction at the positive pole.

In diseased conditions it is necessary to compare the amount of current required to bring about contractions of muscles situated in similar areas on the two sides of the body, or to compare the amount of current required in the affected individual

with the amount required in a healthy individual. Care must always be exercised, however, to exclude local conditions that may interfere with the transmission of the current, such as diseases of the skin, scars, etc.

Simple decrease of electric excitability takes place in atrophy from disuse, in primary muscle affections, such as muscular dystrophy, in atrophy of muscles following diseases of the joints, and in very mild inflammatory conditions of the nerves. Increased reaction to electric stimulation is unusual, but is present in marked degree in tetany.

Myasthenic Reaction.—This reaction has been described by Jolli, and occurs only in myasthenia gravis. It is characterized by the fact that muscles can be stimulated to contraction by the faradic current, but if the faradic carrent is continued for some time, the current being interrupted at intervals, there gradually occurs a decrease in the force of the muscular contraction, until finally the muscles cease to contract even when the current is increased in strength. After a period of rest the muscles will again respond to stimulation.

Myotonic Reaction.—Faradic stimulation of the nerves with a strong current causes tonic muscular contractions that persist after the interruption of the current. Continuous faradic stimulation sometimes causes wave-like contractions of the stimulated muscles; galvanic stimulation of the nerves is somewhat diminished. Direct galvanic excitability of the muscles is increased, contractions occurring only upon closing the current; these contractions are characterized by the slowness with which they occur, their tonic character, and their persistence.

Tetanic Reaction.—Very weak currents produce muscle contractions—this is less constant to the faradic current than to the galvanic. A very weak galvanic current will produce contraction upon closing of the circuit at the negative pole; a slight increase of the strength of the current produces a tetanic contraction of the muscles at the negative pole.

In some cases a tetanic contraction occurs with a weak current at the positive pole upon interrupting the current.

Reaction of Degeneration.—Upon the extent of the involvement of the nerves are dependent the qualitative changes, which

may vary from slight alterations from the normal to a complete reaction of degeneration.

Complete reaction of degeneration is characterized by the following symptoms: (*a*) Faradic stimulation of the affected nerve is not followed by muscular contraction, independent of the strength of the current; (*b*) faradic stimulation of the muscle is not followed by a contraction in spite of the strength of the current. (*c*) Galvanic stimulation of the nerves is not followed by muscle contractions, either upon opening or upon closing the current, and these are not influenced by the strength of the current. These three symptoms are caused by the inability of the affected nerve to transmit the electric current.

Upon galvanic stimulation of the muscles the following alterations from the normal take place: First, the excitability of the muscle is increased; that is, a more marked contraction is brought about in the affected muscles than in the unaffected muscles when currents of equal strength are used, or weaker currents cause the muscles to contract; second, there is an alteration in the way in which the contraction occurs—under these conditions the contractions are slow and wave-like; third, there is a change from the normal in the strength of the contraction to the negative and the positive pole—under these conditions the contraction occurring at the positive pole upon closing the current is stronger than the contraction that occurs at the negative pole upon closing the current.

The exaggeration of the galvanic irritability is found only in the first stages of the disease and gradually diminishes. The most constant symptom in this reaction is the slow, wave-like contraction of the muscle.

In some cases, instead of complete reaction of degeneration there occurs a condition that is characterized by a slight decrease of the irritability of the nerves. In this condition faradic stimulation of the nerves is not followed by muscle contraction, or very strong currents may be followed by muscle contraction—faradic stimulation of muscles is followed by contraction, stronger currents than normal being used. Galvanic stimulation of the nerve is generally not followed by muscle contraction, galvanic stimulation of the muscles upon closing of the current causing

a slow, wave-like contraction that may be more marked at the positive pole than at the negative pole. There are many stages of variation of electric reaction between a mild partial and a complete reaction of degeneration.

In making an electric examination one should compare the reaction obtained in a healthy muscle with that obtained in the muscle under examination. The process of degeneration in the nerves and the muscles occurs more or less rapidly after the onset of a diseased condition. The changes in the electric excitability of the muscles and nerves occur simultaneously with the degenerative changes that take place in the nerves and muscles; consequently, as from a few days to nearly two weeks may elapse before the degenerative changes that have begun in a nerve and muscle are complete, one cannot expect the reaction of degeneration to occur before the end of from a few days to two weeks. In diseased conditions in which there is complete atrophy of muscle and muscle tissue is replaced by fibrous tissue formation all electric excitability is lost.

The reaction of degeneration is present in all severe diseases of the motor or mixed peripheral nerves and in destructive diseases of the anterior horns and anterior roots. Very frequently muscles recover their functions before the electric reactions have returned to the normal.

EXAMINATION OF THE CRANIAL NERVES

The First, or Olfactory Nerve.—This is the special nerve of the sense of smell, and is tested in the following manner: The patient should close his eyes, and the examiner should place near the nostril such non-irritating substances as orange-peel, cinnamon, or a weak solution of the extract of rose or of lavender. Each nostril should be tested separately. As the substance is brought to the nose the patient is to signify whether or not he recognizes or perceives the odor. He should also be questioned as regards the existence of hallucinations of smell.

The Second or Optic Nerve—the Nerve of Vision.—In testing this nerve one should determine whether or not the patient sees, testing each eye separately. The patient's vision should

be tested as regards his ability to differentiate various colors, and the field of vision for white and colors should be taken.

Examination should be made for refractive errors. In all cases a careful ophthalmoscopic examination should be made to determine the condition of the optic nerve and of other structures in the fundus of the eye.

The Third Nerve, or Motor Oculi; the Fourth Nerve, or Patheticus; the Sixth Nerve, or Abducens.—These three nerves supply the various muscles, internal and external, of the eye. They are tested in the following manner: The patient is asked to follow the movements of the examiner's finger, which is moved outward, upward, downward, inward, and then in a circle. In case weakness, paralysis, or disturbance exists, this will be evidenced by the inability of one or both eyes properly to follow the examiner's finger. In making this examination one must be on the alert to detect weakness, paralysis, nystagmus, etc., of the muscles of one or both eyes. If nystagmus is present, the variety, the direction, whether active or passive, must be noted.

Examination of the Pupils of the Eyes.—One must observe whether the pupils are equal in size, whether they are irregular in outline, and their size. The pupillary reactions are tested for in the following way: The patient should be examined in a room that is not too well lighted. He must not face the light, and should fix his gaze upon some object at a distance. The examiner stands at the side of and slightly in front of the patient, and light from a small hand-lamp or some other source is thrown into the pupil. Under normal conditions the pupil promptly becomes smaller; in abnormal conditions the contraction of the pupil may be slow or may not occur at all.

In testing for the pupillary reaction to accommodation one must be able to see the pupils distinctly. The patient is then told to fix his gaze at some distant point; he is next directed to look at the point of a pencil held in the examiner's hand. The pencil is then gradually moved toward the patient's nose, when, as the two eyeballs converge toward the median line, there occurs, under normal conditions, a contraction of the pupils.

Argyll Robertson pupil is one which fails to react to light, but does react to accommodation.

Consensual Reaction.—Here again the examiner must be able clearly to see the two pupils. Upon throwing more light into one pupil, under normal conditions, the opposite pupil decreases in size, and upon diminishing the amount of light entering one pupil, the opposite pupil increases in size.

Sympathetic reaction of the pupil is tested for by slightly irritating the skin of the neck in front of the ear below the angle of the jaw. When this is done under normal conditions, a dilatation of the pupils occurs.

The Fifth or Trifacial Nerve.—The tests applied to ascertain the condition of this nerve include sensory, motor, the special sense of taste, and reflex activity. In testing for the sensory quality of this nerve the examiner should apply the ordinary tests for sensation; that is, tactile, pain, and temperature sense to the skin and to the mucous membranes of the face, side of the head, nose, mouth, etc., in the region supplied by this nerve. If disturbance exists, it should be noted whether the same is limited in distribution to one or more of the principal branches.

The Motor Functions.—The examiner directs the patient to close the jaws tightly and to feel with the fingers whether or not the masseters contract on one or both sides. He is then directed to go through the movements of chewing, in order to learn whether or not the pterygoids are performing their functions.

To test for the *special sense* supplied by this nerve one must examine the anterior two-thirds of the tongue for taste; this is best done by explaining to the patient what is desired, and warning him not to swallow the testing fluids until he has signified whether or not he has perceived the taste. The fluids to be used should consist of solutions of salt, vinegar, and sugar. Let the patient open the mouth fairly wide, and protrude the tongue slightly; then touch one side of the anterior two-thirds of the tongue with one of the solutions, and let him signify whether it is salty, sour, or sweet. Both sides of the anterior two-thirds of the tongue should be tested.

The *reflex activity* of this nerve is to be tested for by the jaw-jerk and conjunctival reflex.

The Seventh or Facial Nerve.—This nerve is to be tested by observing whether the patient can wrinkle the forehead close to

the eyes, draw over the corner of the mouth, smile, whistle, pout, etc. Weakness or paralysis of the muscles supplied by this nerve is indicated by inability to perform these acts equally well on both sides.

The Eighth or Auditory Nerve.—*Auditory Portion.*—In making this test the individual's hearing must be tested with a tuning-fork, watch, etc., both for air and for bone conduction.

Vestibular Portion.—It is also important to test the patient's equilibrium, for in diseased conditions of this nerve rapid changes in position are often attended by a loss of equilibrium, and in some disorders vertigo is more or less constantly present. The patient should always be questioned as to the presence of tinnitus aurium and hallucinations.

Caloric Reaction.—When one ear of a normal person is syringed out with cold water, the head being held upright, a horizontal and rotatory nystagmus toward the other ear occurs; when hot water is used, the nystagmus is directed toward the syringed ear. In cases of destruction of the labyrinth of the vestibular nerve on the side under examination this reaction does not occur.

In special cases the Bárány tests should be carried out in detail.

The Ninth or Glossopharyngeal Nerve.—To test this nerve one should apply the same tests for taste that are recommended in testing the fifth nerve to the posterior third of the tongue.

The Tenth or Pneumogastric Nerve.—In testing this nerve one should test the pharyngeal reflex and the ability of the patient to swallow. He should be given dry and liquid food, and it should be observed whether this is promptly passed into the esophagus or if there is a tendency for it to regurgitate through the nose.

Examination should be made of the larynx to discover whether or not any of the laryngeal muscles are weak or paralyzed. The character of the voice is to be noticed. It must be remembered that this nerve is intimately associated with the action of the heart, the lungs, and the stomach.

The Eleventh or Spinal Accessory Nerve.—This test is made by having the patient turn the head from side to side, flex and extend the head against resistance, and elevate the shoulders.

The state of contraction and the muscular power of the sternocleidomastoid muscle and the upper portion of the trapezius muscle are to be noted.

The Twelfth or Hypoglossal Nerve.—This nerve is tested by examining the state of nutrition of the tongue, and whether there are any abnormalities of movement, and the ability of the patient to move the tongue freely and to protrude the same from the mouth. Weakness or paralysis will evidence itself by interference with the ability to move a part or the entire tongue.

SPINAL PUNCTURE

The withdrawal of cerebrospinal fluid by inserting a hollow needle into the spinal canal is done for diagnostic and therapeutic purposes.

The needle may be inserted into the spinal canal in almost any region of the spinal column, but in some regions this procedure is very difficult, whereas in others it is comparatively simple (Fig. 93).

In making a lumbar puncture the patient may be in the recumbent position lying on his side, or sitting up. In order that this procedure may be readily carried out it is necessary that the spinal column be flexed as much as possible, its convexity being directed backward, thus separating the lamina of the vertebræ. Lumbar puncture was first done by Quincke in 1891.

The needle, which should be about five or six inches long, must be sharp, and contain a stilet; it should not be too flexible. It should be introduced between the second and third or the third and fourth lumbar vertebræ. The needle should be boiled before using, and a sterile dish for the reception of the spinal fluid should be placed conveniently at hand. The patient's back, from the midline to about one and one-half inches on each side, from the ninth or tenth dorsal vertebra to the sacrum, should be rendered surgically clean and aseptic by painting the area with iodin or by thorough scrubbing.

An imaginary line is to be projected across the back at the level of the crests of the iliac bones. The needle is to be inserted at a point on this imaginary line one-half inch on either

side of the midline. It is directed slightly inward and slightly upward. After going through the skin and muscles the stilet should be withdrawn, and the needle gently pushed forward. As soon as it pierces the dura there will be a flow of cerebrospinal fluid.

At times there may be difficulty in entering the spinal canal, as when the needle comes into contact with the laminæ of the vertebræ. Under these conditions the needle should be with-

Fig. 93.—Spinal puncture.

drawn a short distance, and the point either elevated or depressed until it slips between the two laminæ.

In some instances where the needle is inserted too far from the midline, or is directed at an improper angle, it does not enter the canal, but traverses a course forward in the large muscles of the back. In this case it is better to withdraw the needle entirely and make a fresh attempt. If the patient is very restless and resists the effort, or if marked opisthotonos is present, it may be impossible to insert the needle in the lumbar region; in these

cases I have frequently found it possible to perform a spinal puncture in the cervical region, between the sixth and seventh cervical vertebræ, or in the dorsal region. One should find the lower border of the spinous process of the sixth cervical vertebra, and then, after the proper aseptic precautions have been taken, introduce the needle near the midline, below the spinous process, directing it slightly upward.

When the needle is introduced into the spinal canal, under normal conditions the spinal fluid flows out clear at the rate of about 40 to 60 drops a minute. In those conditions in which there is increased intracranial pressure or when an increased amount of cerebrospinal fluid is present, it may flow out in a continuous stream. In cases in which intracranial pressure is decreased and the blood-pressure is very low, where there is evidence of shock, etc., the cerebrospinal fluid flows more slowly than normal. If the needle has been introduced too far into the spinal canal, there usually occurs a puncture of some of the small veins, causing bloody cerebrospinal fluid to appear.

In pathologic conditions the cerebrospinal fluid may be perfectly clear, or it may be turbid, bloody, and purulent. If the cerbrospinal fluid will not run, the patient's head should be elevated, and he should be instructed to take a few deep breaths, which will usually cause a small quantity of fluid to appear.

Where it is desired to measure the pressure under which the cerebrospinal fluid flows, any one of several forms of apparatus may be attached to the needle.

A normal spinal fluid is clear, of a very pale straw color, and contains a few cells and about 0.2 per cent. of albumin. An increase of albumin in the cells and the presence of microörganisms indicate a pathologic condition of the brain and membranes.

CRANIOCEREBRAL TOPOGRAPHY

It is important for surgeons and physicians to be familiar with the relation of the fissures and convolutions of the brain to the surface of the skull.

The frontal bones cover the greater part of the frontal lobes. The base of the frontal convolutions lies beneath the anterior

part of the parietal bones. The precentral and postcentral convolutions, the parietal lobes, and the anterior part of the occipital lobes are situated beneath the parietal bones. The parietal eminence practically corresponds to the upper part of the marginal gyrus. The temporal bones cover the greater part of the temporal lobes. The upper part of the occipital bone covers the occipital lobes, and inferior to the occipital eminence it covers the cerebellum (Fig. 94).

Another important aid is to be able to map out, on the surface of the skull, the fissure of Rolando (Fig. 94). Methods have

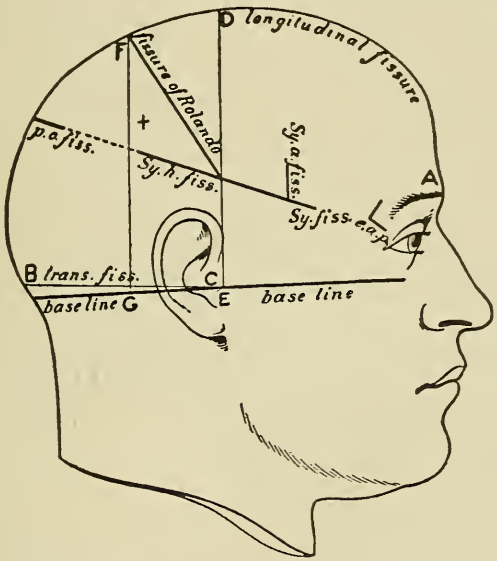


Fig. 94.—Relations of the principal fissures of the cerebrum to the outer surface of the scalp (Reid).

been devised by investigators who agree that the simplest method by which this fissure may be located is to measure the distance from the root of the nose to the external occipital protuberance; this distance is then bisected, and about 2 cm. posterior to this point is the upper end of the fissure of Rolando. From this point the fissure runs downward and forward, forming an angle of about 67 degrees with the sagittal line. The lower third of the fissure of Rolando is somewhat more vertical than the upper two-thirds.

Another method for projecting the fissure of Rolando on the surface of the skull is described by Reid and Bergmann. This is

as follows: At the anterior margin of the external auditory meatus a perpendicular line is drawn from the sagittal line, which is a line from the root of the nose to the external occipital protuberance. About two inches behind this first perpendicular line a second perpendicular line is drawn; this passes across the posterior margin of the mastoid process. At the point at which this second vertical line meets the sagittal line is the upper end

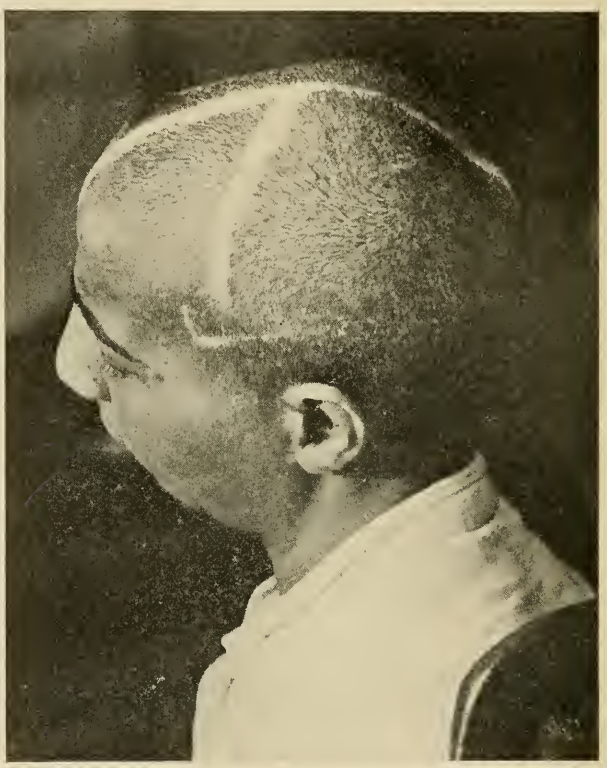


Fig. 95.—Showing fissure of Rolando outlined on the surface of head.

of the fissure of Rolando, the lower end being found on the anterior vertical line, about two inches above the external auditory meatus (Fig. 94).

If one can determine the location of the fissure of Rolando and remember that, approximately, the various lobes of the brain lie beneath the cranial bones of a similar name, it is comparatively easy to determine the relation of the important cerebral centers to the surface of the skull.

DISEASES OF THE PERIPHERAL NERVES

The peripheral nerves, owing to their long course, are frequently injured and exposed to the action of harmful substances that may cause inflammatory changes.

Injury leads, according to its severity, to more or less complete interruption of the conductivity of the affected nerve. It varies from a slight irritation of the nerve that may be either transient or continued, to an injury severe enough to entirely interrupt the conductivity of the nerve.

Injury is brought about by pressure, by section, or by traction. It may be extensive or limited, and may be of momentary duration or continue over a long time. Inflammation of the nerves may arise from the perineurium, and be limited practically to this structure (*perineuritis*); it may be situated mainly in the interstitial tissue (*interstitial neuritis*), or within the nerve-fibers (*parenchymatous neuritis*).

The first two forms correspond to the type of a true inflammatory process. A sharp distinction cannot, however, be made between different forms, as inflammation arising from the perineurium and endoneurium usually involves the parenchyma. The inflammation may be localized or general, ascending or descending, multiple or migratory. After injury or inflammation of a nerve, depending upon the extent and severity, changes take place in the portions of the nerve distal to the involved area and also to a slight extent in that portion of the nerve proximal to the affected area. This process is called the *process of degeneration*, and according to the severity of the lesion do we have partial or complete degeneration. This process was first described by A. V. Waller. It is a change brought about in the part of the nerve distal to the lesion, caused by the inability of the cell to supply nourishment to this portion.

Within from two to four days after the injury the myelin of the nerve becomes disintegrated throughout its entire extent. The

breaking up of the myelin is followed by disintegration of the axis-cylinder, and the nuclei of the sheath of Schwann increase in size and number. The products of this disintegration are gradually absorbed, and the nerve is ultimately replaced by the fibrous tissue cord. The intramuscular ramifications of the nerve and the end-plates also degenerate.

This process does not take place under all conditions, but only when the lesion is extensive and severe, and when regeneration is interfered with. In those cases in which the cause is removed or in which the extent and severity of the injury are not so great, or when the severed ends of the nerves are in good apposition, the process of regeneration takes place. Generally, when regeneration takes place it is due to the outgrowth of new neurofibrillæ from the proximal end of the affected nerve into the remains of the degenerated nerve. There certainly are some undoubted cases on record that seem to prove that it is also possible for the nerves to resume their functions so rapidly that there must be another more rapid process; if this is the case, it occurs only in very few instances. Under favorable conditions the nerve-fibers begin to regenerate within about two weeks after the pathologic process has been removed, and under favorable conditions regeneration takes place at the rate of about one-half inch a week.

If regeneration is prevented, connective-tissue formation occurs in the stump of the affected nerve, and there is an outgrowth of neurofibrillæ, which curl up upon themselves and eventually form a small painful tumor.

If the pathologic process is only very slight and the axis-cylinder is not affected, the functions, as a rule, are rapidly restored, since degeneration does not take place.

The **causes** of diseases of nerves may be grouped under the following heads: Trauma, which may be gradual or sudden, slight or severe, inflicted externally or internally, continued or transitory; toxins, which may be exogenous or endogenous, acute or chronic; chemical agents, climatic conditions, metabolic diseases, hemic conditions, vascular disease; or it may be the result of disease in the neighboring tissue.

Symptoms of disease of the nerves depend upon the extent

and character of the pathologic condition, the character of the nerve or nerves affected, and the distribution of the affected nerve or nerves.

If a mixed nerve is affected, we may have sensory, motor, vasomotor, and trophic symptoms; if the motor nerves are involved, we have symptoms limited to disturbance of function and nourishment of the motor apparatus, and perhaps, also, vasomotor symptoms.

In the case of the sensory nerves, we have disturbance or perversion of sensation, with or without vasomotor and trophic symptoms.

Motor symptoms—either weakness or paralysis; in these cases the muscle tone is decreased, and the paralysis is of the flaccid type. The tendon reflex of the affected muscles is either diminished or lost. The paralysis is followed rapidly by atrophy, except in those cases in which the weakness is limited and the involvement is very slight.

The normal electric reactions are also disturbed. The use of the electric current, galvanic and faradic, plays a very important part in the diagnosis and treatment of diseases of the peripheral nerves. The faradic or interrupted current exerts its effect upon the muscle-fibers by being conducted through these by nerve-fibers. In order to secure the effect of the current it is necessary that intact or practically healthy nerve-fibers be present. The galvanic or continuous current is transmitted both by nerve-fibers and by muscle-fibers, and in diseased conditions, where the nerve-fiber is affected, the muscle-fibers readily convey this current. Under normal conditions, when we apply the faradic current to a nerve-fiber or a muscle, if the current is sufficiently strong, it produces tonic muscular contraction. If a strong galvanic current is applied to a nerve-fiber or muscle, it merely produces muscle contraction upon the closing or opening of the circuit, the flow of current through the muscle having no visible effect. The muscle contraction is sharp and quick, and is more marked at the negative than at the positive pole.

In diseased conditions of the peripheral nerves there are disturbances of the muscle reaction to the electric current. This reaction is known as the reaction of degeneration, and is present

whenever there is an abolition of the conductivity of the nerve, lasting until the nerve either regenerates or all the muscle-fibers have atrophied and have been replaced by fibrous tissue.

In the reaction of degeneration we have the following responses to the current: The application of the faradic current to the nerve or muscle produces absolutely no muscle response, no matter how strong this current may be. On applying the galvanic current we get a muscle contraction that is stronger at the positive pole than at the negative, and stronger upon closing the circuit than upon opening it. The muscle contraction, instead of being sharp and prompt, is slow and wave-like, and the galvanic stimulation through the nerve is lost. Partial reaction of degeneration occurs when a very much stronger faradic current than normal is required to bring about muscle contraction, whereas in direct galvanic stimulation the sluggishness of contraction becomes evident.

Sensory Symptoms.—*Paresthesia.*—Slight involvement of the nerve may give rise merely to symptoms of irritation, tingling, and numbness. If the lesion is relieved, there is a rapid disappearance of these symptoms. A slight general numbness of all the fingers of both hands, and even of the hands and wrists as well, occasionally occurs, and may be of short duration or may become permanent. This is seen chiefly in those who use the hands continuously for hours. It has been called by Dana *acroparesthesia*, and may be attended by a sense of weakness.

Pain.—This may be local, or may follow the distribution of the affected nerve or nerves. It is particularly apt to be present, although it varies in severity, whenever there is irritation of the proximal end of the affected nerve. It usually occurs after injuries in which the nerve is torn or is exposed, or in those cases in which local inflammation exists. In painful conditions the pain is not only felt locally, but is generally referred to the peripheral distribution of the nerve. Pain does not always remain limited to the distribution of the affected nerves, but may extend to neighboring structures.

Tenderness.—This condition frequently accompanies painful states, but may be present even if the pain is not a prominent

symptom. It may be limited to the nerve-trunk or may be diffuse.

Disturbances in the Conduction of Sensation.—There may be a decreased rapidity of the conduction of sensory stimuli, resulting in delayed and consequent slowness of perception, or there may be difficulty in the transmission of the stimuli, whereby the acuteness is decreased and an obtundation or anesthesia results.

After disease or injury of the nerves the individual may rapidly regain the protopathic sense, whereas disturbance of the epicritic sensibility persists for considerable time. In lesions of a single nerve affections of sensibility are usually limited to an area much smaller than would be expected from the anatomic distribution of the nerve.

Vasomotor, Secretory, and Trophic Disorders.—These disorders frequently occur in diseases or injuries of the peripheral nerves. They consist of redness, with local rise of temperature, which may be succeeded later by cyanosis and subnormal temperature. There is increase, decrease, or absence of the perspiration. The skin is smooth and glossy, very thin and tender, and there are superficial ulcerations, herpes, and blisters. The growth of the hair and nails may be affected. When extensive destruction of the nerves close to the spinal column occurs in childhood the bones may cease to grow.

The prominent symptoms of disease of the peripheral nerve, depending upon the severity and the extent of the disease, *are flaccid motor weakness* or *paralysis, loss or decrease of the tendon reflexes, muscular atrophy, partial or complete reaction of degeneration, paresthesia, tenderness, pain, anesthesia,* and *vasomotor, secretory, and trophic disturbances.*

Unless the disease of the nerve is due to some sudden overwhelming condition, the first indications consist usually of paresthesia and hyperesthesia in the area of distribution of the affected nerve. There may also be evidence of slight muscular irritability, such as tonic contractions, fibrillary tremors, or increased electric excitability.

Course and Prognosis.—The onset may be acute, subacute, or chronic. The acute cases are generally secondary to trauma. Subacute and chronic cases are slower in their onset.

In most cases of peripheral nerve affections the prognosis as regards recovery is good. Wherever the causative agent can be removed, recovery should take place, except under the following conditions: If there is a physical impediment to nerve regeneration, such as callus, fibrous tissue, scar formation, or the absence of nerve tissue, or if the nerves are affected very close to their points of emergence from the spinal column.

The *duration of the disease* depends upon the severity, extent of involvement, and the possibility of removal of the cause. In those cases in which the cause can be entirely removed and the electric reactions are normal, recovery should take place within six weeks. In those cases in which the causative agent can be removed and there is partial reaction of degeneration, recovery should take place within from four to six months. When the cause can be removed and there is complete reaction of degeneration, recovery should take place in from six months to two years. The prognosis is, of course, dependent on the ability to remove the cause, the conditions under which treatment is carried out, and on the treatment itself.

Treatment.—The prime requisite for successful treatment is to discover and to remove the cause. If the condition is due to injury, it is necessary to institute proper local treatment. If one has reason to believe that the nerve has been severed and that the ends are displaced, it is my opinion that one is justified in making a surgical exploration to bring the ends of the severed nerve into apposition, suturing them if necessary. In all recent acute cases absolute rest for the affected part is the first indication, every movement and muscle contraction being likely to excite inflammatory processes. All pressure or traction upon the affected nerve should be avoided. The treatment is given in detail in the discussion of the special forms of neuritis.

SPECIAL FORMS OF TRAUMATIC AND INFLAMMATORY DISEASES OF THE SPINAL NERVES

Paralysis of the Phrenic Nerve.—Paralysis of the phrenic nerve is very uncommon because of the protected position of the nerve. It may occasionally, however, be produced by

compression between the clavicle and the spinal column, or as the result of compression by tumors in the neck or throax. The nerve may be involved in toxic or infectious conditions. Diphtheric paralysis frequently involves this nerve.

Paralysis of one side of the diaphragm gives rise to no symptoms, and can be diagnosed only as the result of very careful physical examination.

The *symptoms* of bilateral paralysis of the phrenic nerve are those of paralysis of the diaphragm. The latter does not contract on inspiration, and there is consequently no protrusion of the epigastrium. On expiration the abdomen protrudes and the liver descends. Dyspnea is not always present, but usually occurs upon movement and may be very severe.

The *prognosis* is good in the rheumatic and diphtheric forms. In multiple neuritis and under other conditions involvement of the phrenic nerve has an unfavorable prognosis.

Treatment.—This consists in removing the cause and treating the general condition. Every effort should be made to prevent overexertion and to avoid pulmonary complications. Internally, strychnin in proper doses should be given three or four times a day.

Cervicobrachial Neuritis.—Under certain conditions the nerves comprising the cervical and the brachial plexuses may all be involved, and under other conditions the disease process may involve only a small portion of either one of these plexuses (Fig. 96).

Etiology.—Extensive involvement of these two plexuses rarely occurs. The condition is most prone to affect individuals after the thirtieth year. La grippe is the most frequent of the causative agents, may also follow infectious diseases, extensive injuries, tumors, inflammatory conditions of the neck, tubercular and arthritic conditions of the cervical vertebræ.

The *symptoms* of extensive involvement usually consist of pain extending through the occipital region, shoulder, arm, and forearm; weakness or paralysis of the deep muscles of the neck, shoulder, arm, and forearm; decrease or loss of the triceps, biceps, and periosteoradial reflexes; partial or complete reaction of degeneration of the affected muscles; sensory disturbances,

varying from paresthesia to anesthesia of the neck, shoulder, arm, forearm, and perhaps the hand. Except in very severe involvement of these two plexuses, pain is the most prominent symptom.

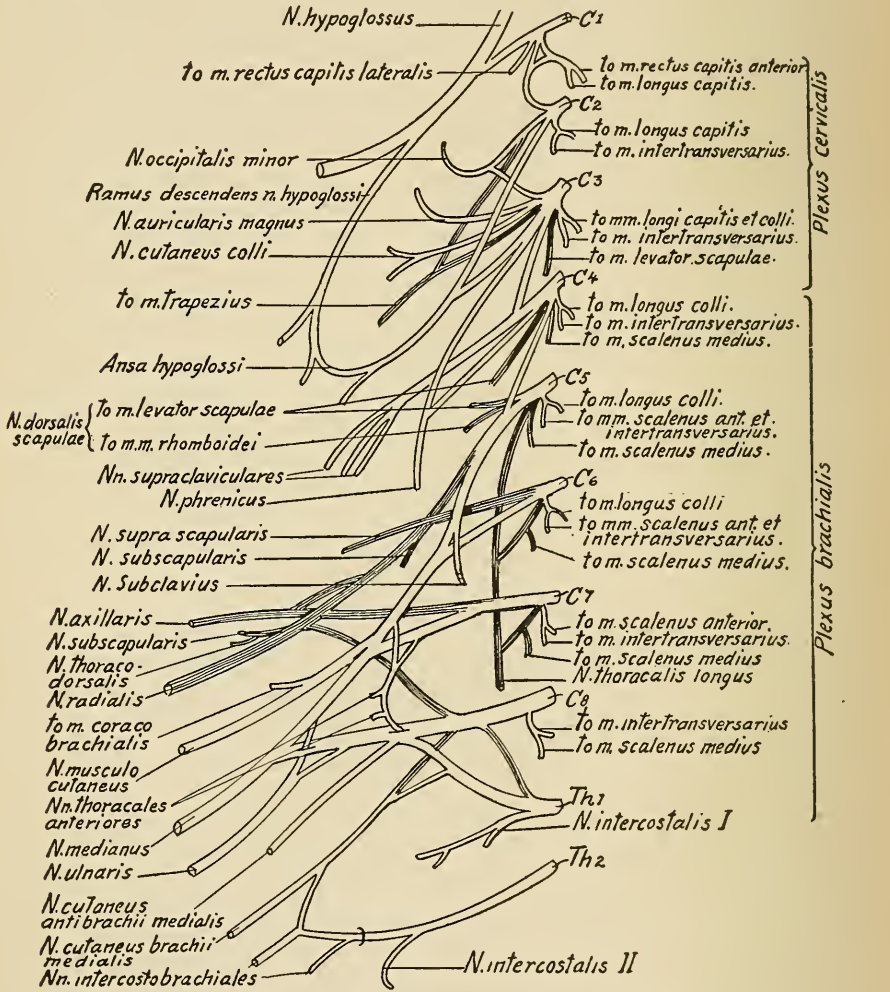


Fig. 96.—Cervicobrachial plexus (after P. Eisler).

The *course* of this condition is usually somewhat prolonged, lasting from three to eighteen months.

The *diagnosis* is made from the history of the onset and the distribution of the symptoms. The most important diagnostic

aid is the discovery of the cause; this often requires a very careful physical examination and the use of *x*-ray.

The *prognosis* depends upon the severity of the case and the ability to remove the cause.

Treatment.—Remove the cause. Put the parts at rest for a short time, remembering that if the shoulder-joint is immobilized for too long a period trophic changes may take place that will later result in limitation of movement. My experience has been that the best results are obtained if passive motion of the shoulder- and elbow-joints is practised throughout the illness, care being taken not to allow the patient to participate in these movements. Warm applications and counterirritation along the side of the neck and over the trapezius muscle are useful in cutting short the acuteness of the condition and in relieving pain. Pyramidon, phenacetin, salicylate of soda, codein sulphate, or morphin may be used for the relief of pain. The positive pole of the continuous galvanic current applied over the affected area with the negative pole at some indifferent point of the body may also give relief. As soon as the condition of the parts will permit, gentle massage is to be practised. The affected muscles are to be treated electrically, using that current which will produce muscle contraction. The patient's general nutrition is to be maintained, and as soon as the acute stage has passed he should be given strychnin sulphate internally in proper doses three or four times a day. In addition to this I have also found small doses of potassium iodid to be of benefit.

The disease may be limited to the cervical plexus, and in this case the symptoms would be limited to pain in the back of the neck, over the occipital region, disinclination to move the head because of the pain, weakness or paralysis of the deep neck muscles, and occasionally trophic disturbances, as evidenced by the falling out of the hair and herpetic eruptions.

Disease of the Brachial Plexus.—This may be caused by injuries to the neck, as from a blow, stabbing or cutting wounds, a fall on the elbow, dislocation of the humerus, fracture of the shoulder-joint or clavicle, pressure on the brachial plexus between the clavicle and the first rib or accessory cervical rib, traction,

etc., tumors pressing on the brachial plexus, rheumatic, toxic, or infectious agents (Fig. 97).

Fig. 97.—Extensive injury to brachial plexus, caused by tree falling on shoulder, causing complete atrophic paralysis, with marked sensory disturbance of entire right upper extremity.

The plexus may be completely or partially involved, the symptoms depending upon the position and extent of the involvement.

One of the commoner forms of partial paralysis is that known as *Erb's combined shoulder-arm paralysis*. This affects the

deltoid, biceps, brachialis anticus, and supinator longus muscles, and sometimes also the supinator brevis, infraspinatus, and subscapularis. This form of paralysis arises from a lesion of the fifth and sixth cervical roots or the upper primary cord of the plexus. It usually is produced by traction, tearing, pressure between the clavicle and first rib, pressure upon the nerves as from carrying weights upon the shoulder; or it may be secondary to some toxic condition or the result of compression by tumors, etc. The symptoms that result consist of inability to abduct the arm or to flex it at the elbow-joint; pronation of the forearm and hand, with the supinating power limited, and some inward rotation of the arm. The arm hangs at the side, pronated and rotated inward. The weakness or paralysis is atrophic, with complete or partial reaction of degeneration, loss of tendon reflex of the supinator and biceps, with retention of the triceps jerk. Pain may be present. Sensory disturbances, if they occur, are usually limited and disappear rapidly. In some cases there may be persistent anesthesia in the cutaneous area innervated by the circumflex and musculocutaneous nerves; that is, in the small area over the shoulder and on the outer side of the upper arm, perhaps on the external surface of the forearm.

Paralysis of the Inferior Plexus.— *Klumpke's paralysis* is due to an affection of the eight cervical and first dorsal roots or the part of the plexus which they form. It may be caused by tumors, surgical operations, gun-shot wounds, traction, or may be secondary to infectious conditions, etc. The symptoms are paralysis of the small muscles of the hand and of some of those of the forearm, especially the flexors (Fig. 98). Sensory disturbances are usually present in the area supplied by the ulnar nerve and sometimes part of the distribution of the median nerve.

The entire brachial plexus may be involved, but this is uncommon. The involvement may at first be general, but may later become limited to the area of certain nerves. The circumflex and musculospiral nerves are most often involved, probably by lesion of the posterior cord of the plexus. Sensory symptoms are usually present, but vary greatly in extent, and no agreement

necessarily exists between the motor and the sensory involvement.

Cervical ribs, by compressing the plexus, may produce symptoms of irritation and paralysis.

The *diagnosis* of disease of the brachial plexus, either in part or in its entirety, is made from the presence of weakness or paralysis of a flaccid character, sensory, vasomotor, and trophic

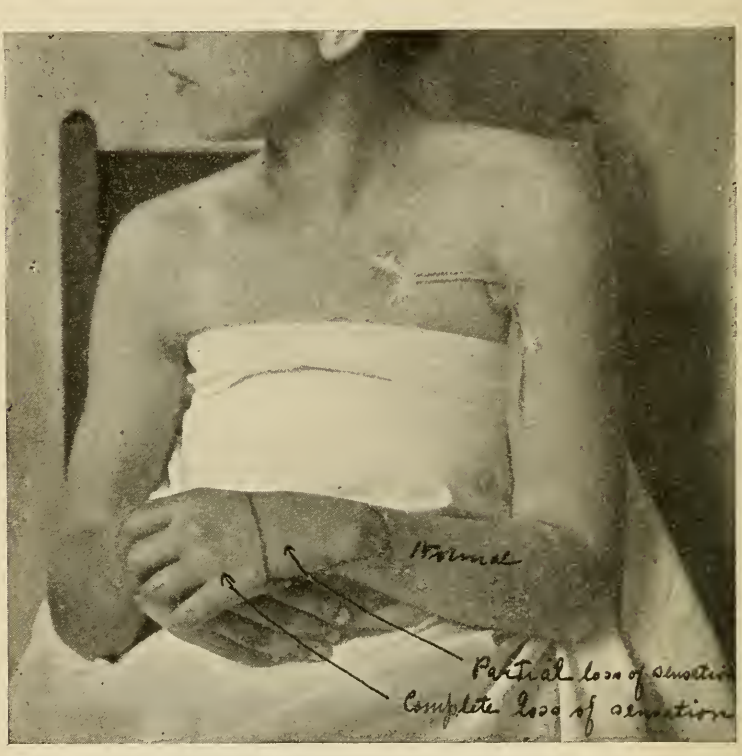


Fig. 98.—Injury of lower brachial plexus, Klumpke's paralysis resulting from gun-shot wound.

disturbances, disturbances of the tendon reflexes and electric reaction occurring in the distribution, which can be referred only to cervical roots, primary trunks of the brachial plexus, or the plexus itself.

The *prognosis* depends entirely upon the possibility of completely removing the cause, and also upon the extent and severity of the involvement of the affected nerves.

In those cases due to traction, if at all severe, due to cutting injuries or secondary to dislocation of the shoulder, the outlook for complete recovery is not good. As a rule, some atrophic paralysis remains, regeneration being very slow.

The *treatment* of diseases of the brachial plexus resolves itself into the treatment and removal of the cause, the relief of pain, and surgical intervention if there is severance of the nerve.

For the relief of pain, aspirin, phenacetin, codein, morphin, etc., in proper doses, may be given. In many cases it may not be necessary to resort to drugs, for the pain can be relieved by hot applications, rest, and the application of the positive pole of the galvanic current to the painful area for from three to five minutes. Every effort should be made to render the patient comfortable.

Trophic disturbances in the distribution of the affected nerves can often be prevented by protecting the parts from pressure or injury and by strict attention to cleanliness. The nourishment of the muscles and skin may be partially maintained by passive exercise and gentle massage with cocoa-butter. As soon as the acute inflammatory stage of the nerves has subsided, and with very few exceptions immediately in traumatic cases, the use of electricity is advisable. The faradic or interrupted galvanic current is to be used. If the faradic current is used, one pole should be placed proximal to the lesion and the other pole applied over the distribution of the affected nerve or nerves. In those cases in which the faradic current fails to excite muscle contraction the interrupted galvanic current will be required. One pole is placed over the muscles and the other on the affected nerve, or both poles may be placed over the muscles, using that pole over the muscle that will give the best muscle contraction with the least current. The current should be automatically interrupted from 60 to 120 times a minute. The faradic current should be applied for about five or ten minutes over the entire affected area. The galvanic stimulation should be applied to each one of the affected muscles for a few seconds, after which they should be allowed to rest and then again be stimulated, giving each muscle about one to two minutes' exercise.

Surgical treatment is demanded in traumatic cases that show no tendency to recovery after three or four months' general treatment.

Brachial neuritis due to exposure to cold is a common disease. Pain localized in the plexus and radiating outward is at times extremely severe. Soon after the onset there are numbness and tingling, followed by pain which is more or less constant, and finally by weakness and paralysis. The onset of the disease covers a period of several days, and within ten days the muscles may show distinct atrophy and the reaction of regeneration.

Trophic disturbances of the skin, such as herpes or eruptions, thin, glossy skin, interference with the growth of the nails, and abnormalities of the secretions are common. The skin is usually hypersensitive and covered with perspiration. These cases generally last for from four to twelve months. The prognosis is good, most cases making a complete recovery.

The *treatment* consists of absolute rest, hot applications, and counterirritation over the affected nerve-trunks, the positive pole of a weak continuous galvanic current being applied to the painful region for several minutes every day. Analgesics should be given for the relief of the pain, and sodium salicylate, aspirin, etc., in proper doses, be frequently administered throughout the day. The bowels should be kept well open, and later, when there is less pain, the use of the interrupted electric current and massage to the affected muscles are indicated. General nutrition should be maintained and as soon as the acute stage has passed, strychnin and other tonics should be prescribed.

Obstetric Paralysis, or Birth Palsy of Duchenne.—Under this head we include only paralysis of the upper extremities occurring in the child (Figs. 99, 100). It is caused by compression or traction exerted against the brachial plexus either by a finger or hook, or by the clavicle when the effort is made to deliver a delayed shoulder. The condition may be caused by the pressure of forceps if the blades reach the cervical region. It occurs also in those cases in which the arm has become extended and the effort is made to bring the arm down; in breech presentation in which there is difficulty in delivering the head and traction is made with pressure against the shoulder.

An obstetric paralysis is seldom seen to occur in deliveries effected without artificial assistance. The commonest type of obstetric palsy is due to an involvement of the fifth and sixth cervical roots, occasioning weakness or paralysis of the deltoid, biceps, brachialis anticus, supinator longus, etc. The arm is rotated inward, the hand pronated, and the forearm extended. Rarely sensory disturbances occur. In a few cases, usually the

Fig. 99.—Position of arm and hand in peripheral type of birth palsy.

effect of violence, the whole plexus may be involved. In such cases there are frequently fracture of the humerus, paralysis of the entire arm, and marked sensory disturbances.

Prognosis.—Obstetric paralysis is very seldom bilateral, and the prognosis is usually favorable. In many cases the paralysis disappears within a few weeks, whereas in others it may exist for several months. In nearly all cases there remains some

weakness which disappears only after years. In a few cases there is no improvement or only partial recovery.

Treatment.—As soon as the condition is noticed, and if there is no surgical contra-indication, such as a fractured humerus, the unaffected arm should be fastened to the body for a part of each

Fig. 100.—*x*-Ray of forearm in same case of obstetric paralysis, showing the retarded development of the bones of the forearm.

day, so as to encourage the use of the affected arm. The arm should be gently massaged daily. Deformity is to be avoided by giving proper support to the arm and hand. Electric treatment should be commenced early, using the current that will cause muscle contraction. Later suitable gymnastic exercises may help many of these cases. If there is no tendency to

recovery, surgical measures should be instituted after the fourth year.

Paralysis Due to Disease of the Long Thoracic Nerve.—The long thoracic nerve may be directly injured in the supraclavicular fossa or in the axilla following injuries to the shoulder, by carrying weights on the shoulder, etc. It affects men more frequently than women. It may also occur after diphtheria or typhoid.

The *symptoms* of disease of this nerve are those of paralysis of the serratus magnus muscle; that is, interference in the power of raising the arm above the horizontal plane, the scapula standing away from the chest. The prognosis depends upon the cause.

Paralysis of the Circumflex Nerves.—This is usually caused by a blow or fall upon the shoulder, compression of the nerve, rheumatic and infectious conditions.

The *symptoms* consist of pain and tenderness over the shoulder, paralysis with atrophy of the deltoid muscle, which prevents abduction of the arm, relaxation of the ligaments of the shoulder-joint, and a small area of anesthesia over the shoulder-joint. This condition must be distinguished from a rheumatic or arthritic condition of the shoulder-joint and also from disease of the deltoid muscle itself.

Paralysis of the Suprascapular Nerve.—Isolated paralysis of the suprascapular nerve is rare. It may occur after dislocation of the humerus or from falls upon the shoulder or hand, causing contusion of the shoulder. The symptoms are paralysis and atrophy of the supra- and infraspinatus muscles. This results in some interference with the rotation of the humerus and with the maintenance of the head of the humerus in close apposition to the articular surface of the scapula.

Paralysis of the Musculospiral Nerve.—The musculospiral is the most frequently paralyzed of all the nerves of the extremities. This is due chiefly to its exposed course and superficial position. It is very easily compressed, resting, as it does, directly on the bone. In the great majority of cases the paralysis is the result of injury or simple pressure during sleep, either from the head resting on the arm or the arm being pressed against something hard. It occurs with especial frequency in alcoholics, and may

follow violent action of the muscles. The nerve may be injured in fracture of the humerus; only very rarely is the condition due to rheumatic influences. It may follow hypodermic injections in the neighborhood of the nerve.

The symptoms are wrist-drop, with pronation of the hand, due to paralysis of the supinators, the extensors of the wrist and hand, etc. The hand is usually flexed at the wrist-joint; the fingers are flexed, and the thumb is in the position of apposition. The hand and the proximal phalanges of the fingers cannot be extended, whereas extension of the other phalanges is not affected. The grasp of the hand is markedly weakened, owing to the abnormal position of the hand and fingers. Some weakness of the power of flexion of the elbow frequently exists.

Paralysis of the supinator longus muscle can readily be demonstrated when the forearm is in a position midway between pronation and supination; forcible flexion against resistance does not cause this muscle to become prominent. The triceps is rarely involved except when the nerve is affected in the axilla, as in dislocation of the humerus or in *crutch paralysis*.

Sensory disturbances are very uncommon, and occur only in most severe cases. The electric reaction depends upon the severity of the case, there being many mild cases that make a rapid recovery and show no electric disturbances. The amount of atrophy also depends upon the severity of the disease. The prognosis is usually very good. Recovery takes place in from a few weeks to several months.

Treatment.—The cause is to be treated and removed. Deformity is to be prevented by properly supporting the hand. Rarely are analgesics necessary. The muscles of the hand and arm are to be massaged daily, and passive and active exercise is to be employed. Electric stimulation of muscles and nerves is useful, the current that will cause muscle contractions being employed. The patient's general condition should be treated, and if no contra-indication exists, strychnin in proper doses should be given. In traumatic cases, if improvement does not occur within from eight to twelve weeks, operative procedures are indicated.

Paralysis of the Median Nerve.—Owing to its protected

position isolated paralysis of the median nerve is an uncommon occurrence, and is usually traumatic in origin. The nerve may be affected along with other brachial nerves in diseases or injuries of the brachial plexus. It may become paralyzed as the result of the use of an Esmarch bandage or following a blow or cut in the upper arm, at the elbow, in its course through the forearm, and near the wrist-joint. It may also be affected in the occupation pareses, in ironers, locksmiths, milkers, cigar-makers, dentists, etc. Puerperal neuritis sometimes involves the median nerve. The median nerve supplies the pronators, the flexors of the fingers, the muscles that abduct, flex, and appose the thumb, and the two radial lumbricales. If the nerve is affected above the wrist-joint, the paralysis is confined to the small muscles of the hand. If the nerve is affected higher up, there is an inability to pronate the hand and to flex the fingers firmly, and the thumb cannot be abducted or apposed to the tips of the fingers.

Sensory disturbances are usually present, and consist of anesthesia of the thumb, first and second finger, and part of the palmar surface of the hand; anesthesia of the terminal phalanges of the first and second fingers posteriorly, and sometimes anesthesia involves the radial half of the ring finger. There is usually some atrophy of the muscles supplied by this nerve.

The electric reactions are disturbed in proportion to the involvement of the nerve. Vasomotor and trophic disturbances occur very frequently in affections of this nerve.

The *course* and *prognosis* depend upon the severity and the cause.

The *treatment* is that usually given for diseases of the peripheral nerve.

Paralysis of the Ulnar Nerve.—This nerve is very frequently injured, as it is exposed at the elbow and in the forearm. It may also be injured along with other nerves of the brachial plexus. Fracture or callus formation, disease of the elbow-joint, pressure, violent flexion of the forearm, prolonged and constant use of the muscles supplied by the ulna, acute infectious diseases, the puerperal state, syphilitic conditions, and exposure to wet and cold may cause paralysis of this nerve.

The ulnar nerve supplies the ulnar flexor of the wrist, the deep flexors of the last three fingers, the adductors of the thumb, the muscles of the hypothenar eminence, the interossei, and the last two lumbricales.

The *symptoms* of ulnar nerve involvement are paralysis of the intrinsic muscles of the hand, bringing about impairment in the use of the thumb and fingers, atrophy of the muscles forming the thenar and hypothenar eminence, and the interossei, giving to the hand a peculiar appearance. There is flatness of the thenar and hypothenar eminence, the bones and long tendons of the fingers stand out beneath the skin, and the little and ring fingers are flexed at the second and third phalanges, with extension of the first phalanges.

Abduction and adduction of the fingers are completely abolished. Sensory symptoms are usually present, and depending upon the severity of involvement of the nerve do we have paresthesia, pain, hyperesthesia, or anesthesia. These disturbances may all be present, or there may be only anesthesia, which is usually limited to the inner half of the hand and little finger and one-half of the ring finger. There may be sensations of cold or heat, vasomotor and trophic disturbances are quite common.

The *course* and *prognosis* are dependent upon the cause and extent of the disease, the prognosis usually being very good.

The *treatment* is that usually applied in diseases of the peripheral nerves.

Neuritis of the Intercostal Nerves.—This is a very rare condition. It may develop in association with herpes, malaria, typhoid, gout, diabetes, alcoholism, and perhaps exposure. The symptoms are pain, tenderness, and sensory disturbances in the distribution of the affected nerve, with atrophy and paralysis of the muscles supplied by that nerve. One should always bear in mind that symptoms that may be referred to disease of the intercostal nerves occur much more frequently in spinal cord conditions.

Diseases of the Nerves of the Lower Extremities.—The lumbar and sacral plexuses are much less frequently affected

than the brachial, and the nerves of the legs are not so often paralyzed as are the nerves of the arm.

Diseases of the Nerves of the Lumbar Plexus.—The lumbar plexus may be affected by tumors or abscesses in the abdomen or by diseases of the spine. It may also be injured in operations performed for the relief of abdominal conditions. The nerves of the lumbar plexus may be independently involved as the result of trauma, chronic intoxication, infectious diseases, etc. The symptoms are usually limited to an area supplied by the individual nerves (Fig. 101).

The **anterior crural** is the branch of the lumbar plexus most frequently involved. The symptoms of involvement of this nerve are weakness or paralysis of the extensors of the leg, atrophy of these muscles, loss of the knee-reflex, pain and tenderness, paresthesia, and sometimes a limited area of anesthesia on the anterior and internal surface of the thigh, in its lower two thirds, on the inner surface of the leg and ankle, and the inner margin of the foot. There is partial or complete reaction of degeneration. If the nerve is injured high in the plexus, there is also paralysis of the muscles that flex the thigh on the trunk.

The **obturator nerve** may also be affected as the result of pressure in difficult labor, pelvic tumors, and in obturator hernia. The symptoms are paralysis of the adductors of the thigh and sensory disorders of the inner aspect of the thigh in its upper third.

Neuritis of the External Cutaneous Nerve.—This condition is also known as meralgia paræsthetica and Bernhardt's disease, and is caused by pressure, overstrain of the legs, infectious diseases, gout, etc. It occurs more frequently in men. It is characterized by paresthesia and pain along the outer side of the thigh. There is usually a small area of diminished sensation of the external surface of the thigh at its inferior part. Standing and walking tend to intensify the symptoms.

The *course* of this condition is generally prolonged. The *treatment* is that which is usually applied in neuritis, special caution being exercised against standing or walking.

Neuritis of the Sacral Plexus.—This affection is very uncommon, and is due to pressure or injury of the plexus during

difficult labor, this being the form of obstetric paralysis that

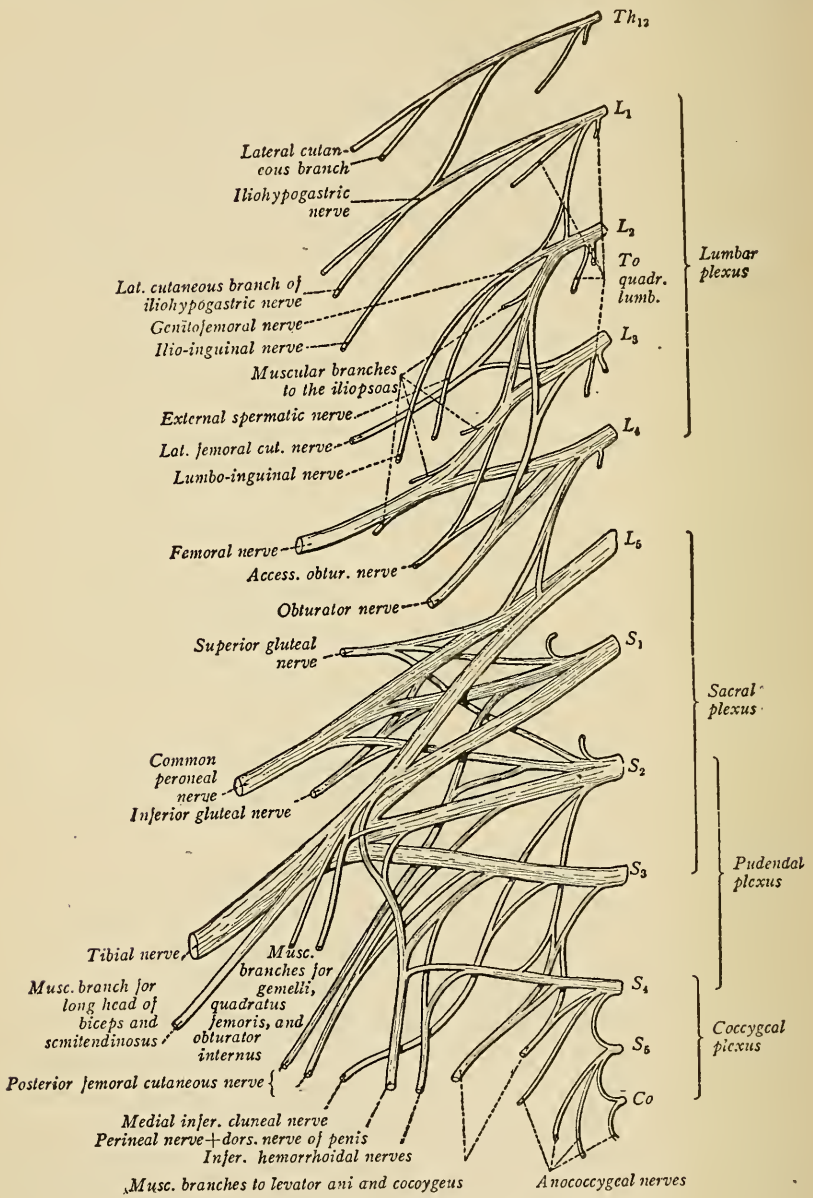


Fig. 101.—Lumbosacral plexus (after P. Eisler).

affects the mother. It may also be due to tumors in the pelvis or to diseased conditions of the sacrum, sacro-iliac disease, etc.

The *symptoms* of a neuritis of the sacral plexus may come on gradually or suddenly, depending upon the cause. There are usually paresthesia and pain in the back of the thigh and down the leg. There may be a flexor spasm of the muscles of the leg. If this condition is more severe, paralysis of the muscles of the leg below the knee occurs, and there are also disturbances of sensation down the back of the thigh and around the outer side of the leg and foot. In milder affections the symptoms disappear within a few weeks; in severer affections the symptoms may require several months to clear up. This condition must be differentiated from diseased conditions of the lower part of the spinal cord: in spinal cord affections the symptoms are usually bilateral, with disturbances of the control of the bladder and rectum; in neuritis of the sacral plexus the symptoms are generally unilateral, and there is no disturbance in the control of the rectum and bladder.

The *prognosis* depends upon the cause, and as this is most commonly difficult labor, the prognosis is good, recovery taking place in from a few weeks to several months. The *treatment* is that which is usually applied in disease of the peripheral nerve.

Neuritis of the Sciatic Nerve.—A true neuritis of the sciatic nerve is a somewhat uncommon condition, although many of the so-called sciatic neuralgias are true neuritic conditions. Neuritis of the sciatic nerve may be caused by injury, pressure in prolonged or difficult labor or during operations for the attempted reduction of congenitally dislocated hip-joint; or it may be secondary to infectious diseases and intoxications, la grippe being a particularly frequent predisposing cause. It often occurs in gouty and anemic conditions.

The *symptoms* are pain in the distribution of the nerve and tenderness along the entire course of the nerve. All movements of the legs are painful, and there is weakness or paralysis of the muscles below the knee, with atrophy and the reaction of degeneration. Vasomotor and trophic disturbances are frequently present. There may be areas of anesthesia along the outer side of the leg and foot. The condition is apt to run a slow course, lasting at times for many months.

Treatment.—Keep the leg and thigh at absolute rest, and

apply counterirritation along the course of the sciatic nerve. Warm applications often give relief; it is advisable to change the position of the affected leg frequently. These patients usually do better if they are kept in bed. Careful examination should be made to discover the cause, and this should, if possible, be removed or treated. Pain is often a very distressing symptom, and efforts should be made to give the patient relief. The positive pole of the galvanic current applied at the upper end of the nerve and the negative pole upon the back may prove helpful. Puncture of the nerve with needles and the injection into the nerve of cocain or of normal salt solution are also used to give relief.

Analgesics, such as aspirin, phenacetin, codein, morphin, etc., are generally required. Gentle massage may be used if it does not cause too severe pain. The use of the electric current and the administration of strychnin internally are to be begun as soon as the patient's condition allows.

Neuritis of the External Popliteal Nerve.—The external popliteal nerve is affected by the same conditions that may involve the sciatic nerve. In addition to these, it may be injured in fractures of the fibula, in pressure caused by tight bandages or garters applied below the knee, or by prolonged compression in extreme flexion of the leg. It may be involved by the extension of inflammatory conditions around the knee-joint, and is frequently involved in toxic conditions.

The *symptoms* of involvement of this nerve are paralysis and atrophy of the muscles situated on the anterior external aspect of the leg and the extensors of the toes, causing dropping and adduction of the foot and toes, known as foot-drop. This gives rise to a peculiarity in walking known as steppage-gait; disturbances of the electric reactions and of sensation on the outer side of the leg and dorsum of the foot also occurring.

The *prognosis* and *treatment* are the same as in affections of the other peripheral nerves.

Neuritis of the Internal Popliteal Nerve.—This is not so frequent as are affections of the external popliteal nerve. The nerve may be compressed or injured, or it may be involved in toxic conditions.

Affections of this nerve give rise to weakness or paralysis of the plantar flexors of the foot and toes, and the individual cannot raise himself on his tiptoes. If the paralysis is extensive, deformity may occur, causing a claw position of the toes, etc. Sensation may be diminished or absent on the postero-external surface of the leg and on the outer margin of the foot, sole of the foot, and plantar surface of the toes.

Prognosis and *treatment* are the same as those in similar affections of the peripheral nerves.

Neuritis of the Plantar Nerves.—This may be caused by injury, such as walking upon very rough ground or the wearing of ill-fitting shoes. It may also result from exposure to wet and cold, etc.

The *symptoms* consist of pain, tenderness to pressure of the nerves between the long bones of the foot, sensory disturbances on the sole of the foot, and paralysis or weakness of the muscles that move the toes.

The *prognosis* is good when the cause can be removed. The *treatment* consists of rest, relief of pain, and other measures recommended for the treatment of neuritis.

Morton's Toe.—This is a painful condition and consists of attacks in which the individual is seized with a sudden sharp pain between the toes. This is so severe as to prevent his taking another step, and it lasts until the shoe is removed, and by manipulation the metatarsal bones are separated. These attacks are likely to occur with great frequency. The condition is due to compression of a digital branch of the plantar nerve in its passage between the ends of the metatarsal bones, and is purely mechanical in origin. It occurs in consequence of wearing shoes that are too narrow.

The *prognosis* is good. The *treatment* is directed toward preventing pressure on the nerve. Shoes should be properly fitted and flat-foot corrected if it exists. A pad should be placed between the metatarsal bones, and if this does not give relief, operative measures must be adopted.

OCCUPATION PARESES

There are certain occupations in which, as the result of pressure upon the nerves or of overuse of a part of the body, there occurs weakness or paralysis, with atrophy and sometimes disturbance of sensation.

These forms of paraylsis occur more frequently in the upper extremities, but have also been known to affect the lower extremities and the trunk. Chronic poisoning and general debilitating states predispose to the condition

In occupations such as those of ironers, file-cutters, locksmiths, carpenters, etc., as result of pressure and overuse, an atrophic paralysis or weakness occurs in the small muscles of the hand. Individuals who use the forearm excessively or lean upon hard surfaces with their elbows, as, for example, telephone operators, glass-workers, oarsmen, etc., are likely to develop ulnar paralysis.

The *course* of the disease is usually chronic; the onset is slow, with a chronic or subacute development.

The *diagnosis* is made by the distribution of the paralysis or weakness, which is flaccid in character, and which may be accompanied by sensory symptoms, and the history of the occupation, which will point to overuse of and pressure upon the nerves supplying the affected muscles.

The *prognosis* is favorable if the cause can be removed.

The *treatment* consists in removing the cause; that is, either entirely giving up the occupation or modifying it in such a way as to remove the possibility of pressure upon the nerves. The general health should be looked after and the affected muscles massaged. The electric currents should be used—the faradic, if it brings about muscle response; should this fail, the interrupted galvanic should be applied.

MULTIPLE NEURITIS (PERIPHERAL NEURITIS)

This condition is characterized by general inflammation or degeneration of the nerves. It is usually more or less symmetric in distribution, appearing upon both sides of the body, and affects most frequently the terminal branches of the nerves.

The varieties of multiple neuritis may be grouped under four heads: First, those cases caused by exogenous toxins, such as alcohol, arsenic, lead, mercury, coal-tar products, carbon monoxid, etc. Second, those cases occurring in the course of infectious diseases, such as diphtheria, la grippe, typhoid, scarlet fever, whooping-cough, erysipelas, septicemia, beriberi, leprosy, etc. Third, those cases occurring in general diseased states of the body, such as rheumatism, gout, anemia, tuberculosis, syphilis, senility, malignant growths, arteriosclerosis, etc. Fourth, those cases following exposure to cold, and which develop without known cause.

Multiple neuritis affects men more frequently than women. It may occur at any age, certain causes acting more frequently in childhood, whereas other causes are more active in young adult life. Probably the greatest number of cases are seen between the ages of thirty and forty years in males. A careful consideration of the histories of many of these cases seems to show that there is usually a complication of different causes which leads to multiple neuritis, such as alcohol and exposure, or alcohol and an attack of la grippe, etc.

The *symptoms* of multiple neuritis, no matter what the cause may be, are more or less similar in the different types. These types are due to the fact that the mode of onset differs in the presence of various causative agents, and these different causative agents seem to exercise a selective activity for certain nerves. We shall first describe the general symptoms of this condition, and then indicate only the variations in different types. There may be a predominance of sensory, motor, or ataxic symptoms. Rarely the symptoms may be limited to disturbances of sensation or motion; usually, however, there is a combination of sensory, motor, vasomotor, and trophic symptoms.

The *sensory symptoms* are generally the first to appear, and begin, as a rule, with paresthesia, such as numbness and tingling. This may be associated with pain of a burning, boring, or tearing character, to which is often added tenderness of the nerves and muscles which may be so extreme as to prevent the handling of the affected parts. These sensory disturbances are very distressing to the patient, and are apt to become more severe as the

disease progresses. Upon examination there may be discovered at first a hyperesthesia, which may later go on to anesthesia. The distribution of these sensory disturbances is usually limited to the distal portions of the extremities—such as are covered by gloves and stockings. Moreover, the distribution of the sensory disturbances is always confined to the areas supplied by the peripheral nerves. In incompletely developed cases there may be loss of one type of sensation with retention of others, such as loss of muscle sense with retention of pain and temperature

Fig. 102.—Foot-drop, multiple neuritis due to alcohol.

sense, or loss of tactile with retention of pain sense. The transmission of sensation is sometimes delayed, but the sensibility to the stimulation is retained. When muscle sense is seriously affected, incoördination and ataxia are prominent symptoms. Sensory symptoms are practically always limited to the extremities, and rarely involve the trunk. The special senses are seldom affected.

Motor Symptoms.—These generally manifest themselves at first by a weakness which gradually extends and becomes more

marked until complete paralysis develops. It may come on within a few days, or may gradually extend over a period of several months. It may cease to advance at any time in its development. This weakness generally begins in the distal portion of an arm or leg, and usually involves both sides at the same time, being, as a rule, more marked on one side than on the other. The muscles supplied by the external popliteal nerve and those supplied by the radial are always more severely affected than are those supplied by the internal popliteal and the median and ulnar nerves. Consequently early in the disease we have greater weakness of the flexors of the ankles and extensors of the wrist than of the extensors of the ankle and flexors of the wrist, this giving rise to foot-drop (Fig. 102) and wrist-drop.

As the disease advances there is an increase of paralysis, which extends to the posterior leg group of muscles, and may involve the thigh muscles, the flexor muscles of the wrist, and the muscles which flex and extend the forearm. In unusual cases the muscles of the trunk, respiration, and the cranial nerves may also become affected. The affected muscles show more or less atrophy, and always lose their normal tone and become relaxed and flabby.

The tendon-reflexes may be somewhat increased in the very earliest stage of the disease, but after destructive changes have taken place in the nerves there is always a loss of the tendon-reflexes.

Electric examination usually reveals the fact that there is a disturbance of the normal reactivity, partial reaction of degeneration being present at times, and complete reaction of degeneration at others.

Vasomotor and Trophic Symptoms.—These are not so constant as are disturbances of sensation and motion. In some cases the affected parts are covered by a profuse perspiration; in other cases the skin may be glossy and appear to be very thin. The nails may become thick and ridged, and there may be an edema of the affected part. The involved area may present a bluish, swollen appearance or be pale and shrunken.

There may be general constitutional symptoms; these are particularly apt to occur at the onset of the disease, when the

patient may have a rise of temperature for several days or weeks, a general feeling of malaise, gastric disturbance, vomiting, constipation, headache, etc. Increased rapidity of the pulse is very common, and may persist during the entire course of the disease.

Course and Prognosis.—These depend upon the cause and the possibility of its removal and upon the severity of the case. Some cases recover in a few months, some last as long as two years, and some terminate fatally.

Differential Diagnosis.—This disease must be differentiated from anterior poliomyelitis, locomotor ataxia, and diffuse myelitis. (See p. 211.)

Treatment.—In treating these cases we must first make every effort to remove and treat the causative agent. This, of course, can be readily done when the disease is caused by a known exogenous toxin. In all cases it is advisable to put the patient to bed, and to give salines to produce free purgation. Water and hot drinks should be taken to assist in elimination. In the early stages of the disease the salicylates, aspirin, pyramidon, etc., seem to have a good effect. Gastro-intestinal disorders, cardiac disturbances, fever, etc., all require proper attention. Pain and tenderness are frequently very distressing symptoms, and every effort should be made to relieve them. The pain can sometimes be relieved by the coal-tar products; at other times codein and morphin must be used. Pain may also be relieved by hot and cold applications and by light massage. The tenderness may be so excessive as to prohibit the slightest touch, and under these conditions special contrivances must be arranged to prevent the pressure of the bed-clothes. Deformity is prevented as far as possible by the arrangement of pillows, etc., to obviate improper positions of the affected parts. As soon as the acute stage has passed, strychnin and the hypophosphites should be administered. In those cases in which we believe syphilis plays a part, mercurial inunctions and potassium iodid should be used in conjunction with the strychnin. In those cases in which malaria seems to play a rôle quinin should be used in combination with the strychnin. If the patient seems to be anemic, iron preparations should be given. As soon as the condition

	Anterior Poliomyelitis.	Locomotor Ataxia.	Diffuse Myelitis.	Multiple Neuritis.
Onset and course.	Usually sudden; reaches its height in a short time; gets better rapidly and then slowly improves for a long period up to a certain point.	Usually very slow and gradually progressive, with intervals of almost complete absence of symptoms.	Slowly progressive.	Onset gradual, extending over a few weeks or months, reaching its height and then gradually improving, to end in recovery.
Age of onset.	Usually in childhood.	Between the thirtieth and fiftieth year.	.....	Except in diphtheric cases, usually between the twenty-fifth and fiftieth years.
Distribution of sensory disturbances.	Usually none present. May be diffuse pain.	Pain, shooting and severe in character, in distribution of segments on cord.	In distribution of spinal segments, gradually advancing upward.	In distribution of peripheral nerves; glove- and stocking-shaped distribution.
Distribution of motor disturbances.	Paralysis and atrophy of muscles which are controlled by spinal cord segments.	Paralysis rarely occurring except in the terminal stage. Transient paralysis.	Paralysis in the distribution of spinal segments, gradually extending from the legs to the thighs and from the thighs to the trunk, and from the trunk to the arms.	Paralysis in the disturbance of the peripheral nerves usually begins in the muscles supplied by the anterior tibial, then involving those supplied by the musculospiral, and as the disease advances gradually involving others of the lower and upper extremities, rarely those of the trunk.
Character of sensory disturbances.	Early in the disease there may be pain in the affected region. This rapidly disappears, and there are no prominent sensory disturbances.	Sharp shooting pains, the girdle sensation, irregular areas of anesthesia on the extremities and trunk, and generally loss of muscle sense in extremities.	Loss or decrease of all forms of sensation extending on the trunk to a point corresponding to the upper limit of the spinal cord involvement.	More marked disturbances of the epicritic than of the protopathic sensation; marked tenderness of the nerves and muscles to pressure; pain more or less constant and very severe.
Rectal and vesical disturbances.	Rarely occurring and then only transient.	Frequently occurring.	Frequently occur.	Never occur.
Trophic disturbances.	Atrophy of muscles; incomplete growth of all tissues of the affected part.	Perforating ulcers and Charcot joints, etc.	Bed-sores.	Glossy skin, ridging of nails, herpes, etc.
Electric reactions.	Disturbances of the faradic and galvanic response in proportion to the amount of muscle tissue lost.	Normal reaction.	Disturbance in proportion to the amount of muscle tissue lost.	Partial or complete reaction of degeneration.
Pupillary disturbances.	None.	Generally present.	None.	None.
Cranial nerve involvement.	Rarely, except in cephalic cases.	Frequently transient cranial nerve paralysis.	None.	Occurring very rarely, except in cases due to diphtheria.

will permit, massage and passive movements should be actively employed daily, and warm baths, packs, and douches should be used to assist in stimulating the circulation and thereby increasing the nutrition and assisting in the reparative process. Electricity is one of the most useful means at our disposal for assisting the progress of nerve regeneration and maintaining muscle nutrition.

The two electrodes should be covered with clean gauze, and be about one and one-half to two inches in diameter. The current which should be used is that which will bring about contraction of the muscles that are being treated. In cases of partial reaction of degeneration I find it advisable to use both the faradic and the interrupted galvanic currents. In very mild cases the faradic current suffices; in more serious cases at first I use only the interrupted galvanic current in the more severely affected areas, and both currents in those areas not so markedly affected. The pole that brings about the best contraction is the one which is to be applied to the affected area. In using the interrupted galvanic current there should be from 60 to 80 interruptions a minute; this is best attained by using an automatic rheotome.

Electricity is beneficial in that it stimulates the regeneration of the nerve-fibers, and, by producing muscle contraction, promotes circulation in the muscle, stimulates its nutrition, and by exercising the function of the muscle, preserves its normal irritability. In applying the faradic current one pole should be placed over the main trunks of the nerve and the other moved backward and forward on the muscles and skin of the affected part. In applying the galvanic current the same method should be pursued as in using the faradic current. When however, we seek to exercise the muscle by the employment of the interrupted galvanic current, the two poles should be placed over or close to the opposite ends of the muscle or muscles that are being treated. The strength of the current should only be such as will bring about contraction.

Contractures should be avoided, but if they have occurred, may be overcome by massage; if this does not suffice, they should be rectified surgically. Where there is anesthesia, the patient should be guarded against injury, and where trophic

disturbances exist, they should receive proper attention. The patient should be encouraged to make attempts at voluntary movements. His general nutrition should be correctly supervised. In these cases the results are most satisfactory if we can eliminate the cause. Active treatment certainly shortens the duration of the disease and prevents troublesome sequelæ.

SPECIAL FORMS OF MULTIPLE NEURITIS

The description just outlined will apply fairly well to all forms of multiple neuritis, there being only a few peculiarities of development and distribution that are more characteristic in one form than in another.

Multiple Neuritis Due to Alcohol.—This is the most common of all forms of multiple neuritis. Although the condition is seen more frequently in men than in women, when one takes into consideration the greater frequency with which men are exposed to intoxication, it will be seen that it occurs more often in women. It attacks by preference those who use beverages in which the alcoholic content is high, such as brandy, whisky, gin, rum, etc., but it may also occur in those who use large quantities of beer and wine. It is more likely to follow the prolonged and constant use of alcoholic liquors, rather than the occasional overindulgence. All ages may be affected, but it is most common between the thirtieth and fiftieth years.

The onset is usually gradual, and the patient may complain for weeks or months of gastro-intestinal symptoms, neuralgic pains, cramp-like pains in the legs, general weakness, and trembling, when suddenly the legs may give way and the patient fall and be unable to rise. From this time on the symptoms are characteristic: there may be a preponderance of sensory or motor symptoms, but usually there is a combination of both. There is weakness or paralysis of the flexors of the ankle, bringing about foot-drop; this is followed by weakness of the calf muscles and weakness or paralysis of the extensors of the wrist, causing the grasp of the hands to be impaired and perhaps resulting in wrist-drop. The weakness or paralysis is flaccid in type. There is usually more or less atrophy of the involved muscles, with loss of the tendon-reflexes and disturbance of the electric reactions.

This paralysis and weakness may extend until practically all the muscles of the four extremities are affected, and in rare cases the cranial nerves may also be involved. The nerves are usually sensitive to pressure, and there is marked muscular tenderness.

The sensory symptoms generally precede the motor symptoms for some time, although this is not invariably the case. At first there may be numbness, tingling, and burning in the legs and feet, followed by pain of quite a severe character and hyperesthesia. The pain and paresthesia, which may at first have been intermittent and limited to the legs and feet, become more constant and more extensive in distribution. As the disease advances, in spite of the fact that pain may continue, examination will reveal delayed transmission of sensation, loss of epicritic sensibility, and finally complete anesthesia. These affect at first small areas limited to the distribution of the terminal filaments of the anterior and posterior tibial nerves, but finally take the form of stocking- and glove-shaped areas. Vasomotor and trophic disturbances occur frequently, manifesting themselves in a profuse perspiration, a bluish appearance, edema of the terminal portions of the extremities, etc.

In some cases marked mental disturbances appear. In many alcoholics delirium, confusion, a stuporous condition, or transitory hallucination occur. In conjunction with these there may occur a mental state known as Korsakoff's syndrome. This is characterized by the symptoms of a multiple neuritis plus disturbances of memory for recent events, and frequently disorientation for time, place, and person. In this condition the patient, although he may be able to recall events that have taken place many years previously, is unable to remember incidents that have taken place within the present, being unable to recall happenings within the space of a few minutes. In addition to this he is frequently unable to recognize his surroundings, and may believe himself to be at home when he is in the hospital. He is unable to recognize those about him, addressing the doctors and nurses by the names of his acquaintances. He is also unable to realize the passage of time, asserting that he has been in the hospital for a day when in reality he has been there for several months. Very frequently these lapses of

memory are interwoven with fabrication, the patient describing in greatest detail events in which he imagines himself to have participated, but which have never taken place. As an example, a patient who has been confined to bed for weeks in a hospital will describe in detail his experience at a theater or party on the preceding evening.

There is one type of alcoholic neuritis in which the loss of muscle sense is the most marked symptom. In these cases ataxia is more prominent than the paralysis, and incoördination is the symptom which first and most persistently attracts attention. This group of cases may readily be mistaken for locomotor ataxia, and the condition is called *pseudotabes alcoholica*. The gait in this class of cases differs from that of the true tabetic in that the latter throws the foot forward, lifting the toes high in the air, and bringing first the heel down forcibly and then the entire foot. The alcoholic, owing to the weakness of the muscles of extension, cannot raise the toes; he therefore lifts the foot high in order to step over the hanging toes, and then throws the leg forward so as to throw up the toes to get them out of the way, and then bringing the foot down on the floor. This is an awkward form of the steppage-gait. In addition to the disturbance of the muscle sense there are generally symptoms of motor involvement.

The alcoholic form of multiple neuritis is characterized by the general constitutional symptoms of chronic alcoholism, by the fact that it usually begins as an affection of the anterior tibial nerves, in which both sensory and motor symptoms are prominent, by plantar hyperesthesia, and by the development, in a few cases, of Korsakoff's syndrome.

Course and Prognosis.—Alcoholic multiple neuritis is usually a subacute condition, and lasts from a few months to two years. In those cases in which Korsakoff's syndrome is present the symptoms of neuritis may clear up before the mental manifestation disappears. As a rule, the mental disturbance does not clear up entirely, there usually remaining a permanent disturbance of the memory.

Korsakoff's syndrome may terminate in complete recovery, in partial recovery, or in marked dementia.

Treatment.—The great difficulty in treating this class of cases lies in the fact that it is very difficult to prevent a chronic alcoholic from obtaining alcohol. In spite of the fact that the patients know the danger of using this drug, they will get it if possible. In the treatment of these cases one must always take into consideration the general condition of the patient, as it may be very unsafe to withhold alcohol suddenly from an individual who is bordering on delirium tremens; under these conditions it is gradually reduced.

Where there are much restlessness, tremor, gastro-intestinal disturbances, etc., the following preparation seems to be of great help. The doses must be regulated according to the age and condition of the patient. If the patient is unable to sleep, chloral hydrate may be added—from 10 to 20 grains being added to the afternoon and evening doses:

℞. Potass. brom. ..... gr. xv;
Tinct. nucis vomicæ } ..... āā ♏x;
Tinct. gentian comp. }
Oleum capsici ..... gtt. ss;
Ess. pepsin. ..... ℥j.

The above is one dose, and is to be given in water every two or three hours, the interval to be lengthened as the patient improves.

The bowels must be freely moved by salines, and the patient encouraged to drink water freely. Pain should be relieved and deformity prevented. Careful nursing, pleasant and bright surroundings, massage, electricity, and strychnin in suitable doses, constitute the treatment.

Arsenical Neuritis—Multiple Neuritis Due to Arsenic.—This condition may occur from the absorption of arsenic or of any of its salts. Arsenic may be taken into the system through the gastro-intestinal tract, either accidentally or with suicidal intent. It may also be taken into the system during the manufacture of certain substances, as in tin workers and in employes of dyeing establishments, sulphuric acid factories, etc. It may be taken into the respiratory tract from the inhalation of particles of arsenic in the air given off from wall-papers, carpets, etc., or chronic arsenic poisoning may occur from the use of toilet

powder that contains arsenic. It may also follow the prolonged use of arsenic for medicinal purposes. The symptoms of arsenical neuritis are those already described. There are certain constitutional symptoms that generally appear before the symptoms of neuritis manifest themselves. If the poisoning is acute, the symptoms consist of vomiting, diarrhea, and abdominal pain. In chronic arsenic poisoning there is a puffiness of the eyelids and gastro-intestinal disturbances, abdominal pain, nausea, greenish, mucous stools, and hyperesthesia occur. In arsenical neuritis, as in alcoholic neuritis, the first symptoms complained of are usually tingling, numbness, and burning in the hands and feet, hyperesthesia of the skin and muscles, and severe pain, described by the patient as of a tearing, shooting nature. These symptoms are accompanied or followed by weakness and finally paralysis. The distribution of the paralysis is always bilateral and symmetric. In some cases ataxic symptoms may predominate at the onset of the disease, but sooner or later the paralytic symptoms develop. This form of multiple neuritis is characterized by extreme pain and tenderness. The cranial nerves, trunk, and diaphragm escape. There may also be trophic disturbances, such as redness, swelling, and profuse perspiration, etc. The skin gradually becomes darker from the deposit of pigment, presenting a mottled appearance, and in extreme cases it turns almost black. The mucous membranes are never discolored. Changes in the nails are also very common—they become thick and brittle, with transverse, roughened ridges. Recovery usually takes place if the patient does not die from the effects of the arsenic poisoning.

Treatment consists on the removal of the cause, and is similar to that usually prescribed for multiple neuritis.

Multiple Neuritis Due to Poisoning by Lead.—This form of neuritis is caused either by the absorption of lead through the gastro-intestinal tract, which is particularly apt to occur in individuals who work in lead and who do not thoroughly wash their hands before eating, or it may enter the system through the respiratory tract. Those who work in badly ventilated establishments, where lead or its salts are used in a dry form, are prone to be affected. In most cases the neuritis is preceded by one or

more attacks of lead-colic, the patient suffering from abdominal pain, nausea, vomiting, and marked constipation. The patient is usually anemic, and there may be a characteristic blue line along the gums. A few days, or sometimes several weeks, after the attack has subsided the patient is seized with weakness or paralysis of the extensors of the wrist and fingers, giving rise to a very characteristic wrist-drop (Fig. 103). In addition to the paralysis the patient may complain of severe muscular cramps.

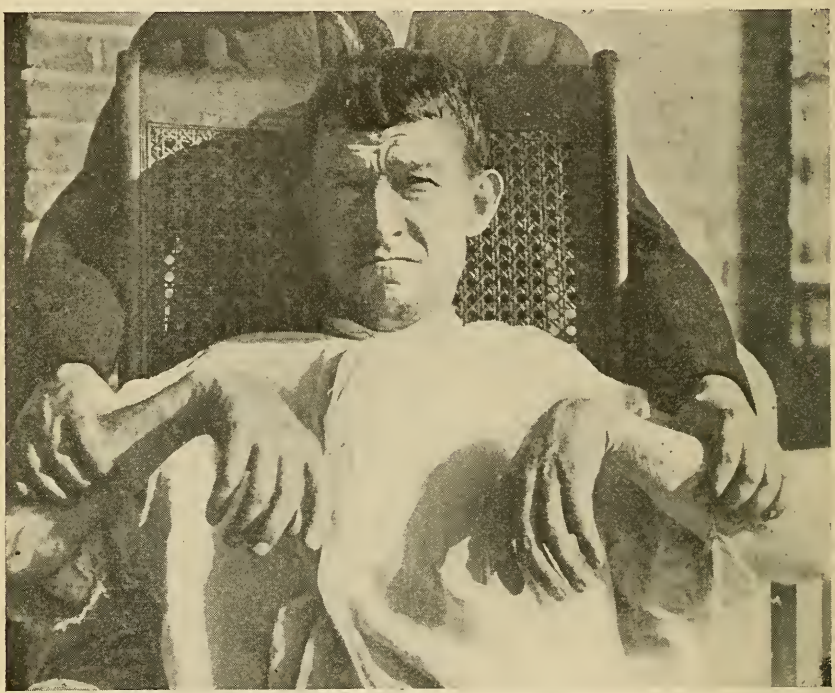


Fig. 103.—Wrist-drop in a case of multiple neuritis due to lead poisoning.

It is only in the markedly severe cases that the paralysis extends to the muscles of the arms and of the legs (Fig. 104). Usually there is little or no tenderness along the course of the nerves. There is also a sensory form, in which the patient suffers intense pains in the arms and legs, with anesthesia of the hands and feet. Another variety, which is rare, is called the cephalic form; in this type there are involvement of the cranial nerves, headache, vertigo, and marked mental dulness. The blood is poor in

quality, and shows basophilic degeneration of the red corpuscles.

The *prognosis* is good in the milder cases, but in the cephalic variety it is very grave.

Multiple Neuritis Due to Illuminating Gas, Coal-tar Products, etc.—Mild forms of multiple neuritis may follow poisoning by illuminating gas and coal-tar products, such as sulphonal,

Fig. 104.—Multiple neuritis due to lead poisoning, severe case in which the paralysis and atrophy involved the muscles of the arms, shoulder, legs, etc.

trional, etc. The symptoms consist of tingling and numbness with some weakness of the extremities, which may persist for weeks or months, gradually subsiding under tonic treatment.

Postdiphtheric Neuritis.—This form of neuritis was formerly one of the most common varieties encountered, but since the discovery of antitoxin it has become infrequent. Nevertheless, multiple neuritis is encountered in children who give a history

of cold or sore throat several weeks preceding the onset of the illness. There is no question in my mind that in these cases a true diphtheric condition has been overlooked, and it is only when the symptoms of neuritis make their appearance that the true nature of the disease is recognized.

The *symptoms* may be limited to difficulty in swallowing and in speaking, regurgitation of fluids through the nose, and there may also occur paralysis of the ocular muscles. In nearly all cases there is a loss of the knee reflex. It has been my experience that in those cases in which the local disease has not been recognized, and consequently has not been treated, if a neuritis develops, the extremities are generally affected, along with involvement of the ninth and tenth cranial nerves. In the more severe cases there is loss of the tendon-reflexes, a tendency to steppage-gait, with weakness of the flexors of the ankle-joints, some disturbance of the muscle sense, and delayed transmission of sensation. In the very severe cases there is great difficulty in swallowing, and weakness and atrophy of the tongue, paralysis of the vocal cords, and marked disturbances in the respiratory and cardiac rhythm. Diphtheric paralysis may affect either adults or children.

The *prognosis* is good, provided there is no involvement of the respiratory muscles or pneumogastric nerve, and even in these cases, with very careful nursing, recovery is possible.

The treatment is that of multiple neuritis, keeping in mind the possibility of respiratory and cardiac paralysis and the danger of inhalation of food. The patient should be kept absolutely quiet until all the symptoms have disappeared.

Puerperal Neuritis.—This type of neuritis may occur during pregnancy or after labor. It is due to a toxemia of unknown origin, and does not differ from the usual form of multiple neuritis. In some cases the sensory symptoms predominate, and in others the motor symptoms, but usually there is a combination of both. The symptomatology resembles closely that of alcoholic neuritis, and in some cases there is also Korsakoff's syndrome. The prognosis in these cases is usually favorable.

Beriberi.—This disease is characterized by certain constitutional symptoms together with the manifestation of mul-

tiple neuritis. There are two theories as to its causation—one, that it is due to an infection, and the other, that it is caused by a toxic substance, such as exists in improperly prepared rice. It occurs more frequently in males than in females, and is most prevalent between the ages of sixteen and twenty-five. Exposure to damp and cold and crowded dwellings increase the susceptibility to the affection. It develops most frequently during the hot damp months of the year.

Symptoms.—The onset is generally gradual, beginning with moderate fever, coryza, conjunctivitis, loss of appetite, nausea, vomiting, a weak, heavy feeling in the legs, and rapid fatigue. The fatigue is soon associated with numbness and pain in the legs, and about this time the patient complains of palpitation of the heart and tenderness of the muscles. The heart may be irregular and rapid, and there may be edema of the extremities. The condition may not progress beyond this point, and the patient may recover in from a few weeks to several months. The disease may, however, be more severe, and present three different types: The first is the atrophic or dry type; in this the weakness develops into a true paralysis, associated with marked atrophy of the muscles. Within a short time the paralysis spreads from the legs to the arms, and may involve the trunk and even the face. The entire muscular system wastes away. To these symptoms are usually added sensory disturbances, paresthesia, general muscular tenderness, and extreme pain; anesthesia, when present, is limited to the extremities. Vasomotor and trophic disturbances frequently occur. The majority of these cases recover, but convalescence continues for a year or more.

The second is the hydropic or wet type; in these cases heart failure appears early, and there is edema of the entire body. The swelling conceals the atrophy, whose presence is indicated by the fact that the paralysis is as severe as in the preceding form. In this variety the prognosis is bad, although recovery may take place.

The third is the acute pernicious type. In this class of cases the symptoms consist of rapid onset of paralysis, edema, gastrointestinal disturbances, and suppression of urine. The pulse

becomes rapid and irregular. This type of the disease may run its course to a fatal termination in a very short time.

Treatment.—Change of climate, if possible, proper attention to the diet, and the relief of the constitutional symptoms are indicated. The treatment of the general condition is most important; as this improves, the treatment is directed toward the relief and cure of the neuritis.

Leprous Neuritis.—This form of neuritis is due to the presence of the lepra bacillus in the interstitial tissue of the nerve-trunks, where, by direct irritation, it produces an overgrowth of the connective tissue, which in turn causes pressure on the nerve-fibers and a secondary degeneration. Small bulbous enlargements are found along the course of the affected nerve.

The *symptoms* consist of the gradual development of irregular areas of anesthesia, the occurrence of paralysis with atrophy, and the production of trophic disorders. The anesthesia is extremely irregular in its distribution, and may occur on the extremities, trunk, or face. The paralysis usually affects the small muscles of the hands and feet, and may extend to the larger muscles of the extremities; not uncommonly the facial muscles are involved. The trophic disturbances which are present, such as the whitish areas on the skin, the peculiar thickening or atrophy of the dermis, the ulcerations, etc., are to be regarded as a part of the disease called leprosy, rather than as the evidences of local affection of the nerves.

The *prognosis* is very unfavorable, and *treatment* is unsatisfactory.

HERPES ZOSTER

This condition is characterized by an eruption of vesicles, which is preceded, accompanied, or followed by pain, more or less severe. The eruption and the pain follow the course of a nerve. This condition affects by preference the trunk, in the region of the dorsal roots, and extends in the form of a half-girdle. The trigeminal nerve is also often affected, particularly its supra-orbital division. Herpes may occur along the distribution of any one or of several nerves. In the great majority

of cases the disease is limited to one side of the body, but a bilateral distribution has been observed in a few cases.

Etiology.—Herpes may be due to a toxemia, not infrequently it occurs in the course of diseases of the lungs, pleura, or gastro-intestinal tract. It may also be due to gout or to injury affecting the nerves or nerve-roots, or it may be a primary acute disease.

Pathology.—Examination of the nervous system generally reveals an acute hemorrhagic inflammation of the ganglia and

Fig. 105.—Herpes zoster; upper eyelid and supraorbital region. (Mraček, "Atlas of Diseases of Skin.")

of the nerves supplying the affected region. The affection tends to be limited—frequently only one ganglion is involved. The disease has also been attributed to a peripheral neuritis.

Examination of the ganglia of the posterior roots and of the sympathetic system shows hemorrhagic and red-cell infiltration, usually more marked just between and outside the capsule and surrounding areolar tissue, and also along the sheath of the nerves supplying the affected area. The blood-vessels show marked congestion. Gram-positive diplococci have been found quite constantly in areas at the seat of lesions and in the

ganglia. From this it would appear that herpes is due to an infection with some form of bacteria that possess an elective affinity for the ganglia on the posterior roots of the spinal nerves or on the cranial nerves.

Symptoms.—The disease is characterized by an eruption of groups of vesicles that appear on a reddened base (Fig. 105). The vesicles contain at first a clear serum, which later becomes opaque and purulent. They then dry up, and brownish crusts or scabs appear which fall off and leave brown spots, which also finally disappear. The number of vesicles and of groups varies. When the eruption is close, the vesicles may become confluent and form one large blister. The development of the eruption usually occupies a period of several days, one group following another. It tends to heal, and the entire process extends over a period of from two or three weeks to two months.

Pain is usually an accompaniment of this condition. It may be very severe and of a burning character, or it may be more moderate in degree. It usually precedes the appearance of the eruption, and persists for a few days after its appearance; it may, however, continue throughout the course of the disease. Sensory disorders may occur, but are uncommon. The temperature is slightly elevated, and general constitutional disturbances are frequently present.

The **prognosis** is usually favorable. The pain may last for a considerable time after the herpes has disappeared. This may prove serious in old cachectic patients.

Treatment should be directed toward the constitutional disease which may exist. The vesicles should not be opened, but should be well covered with carbolized zinc ointment. Friction should be prevented by proper bandaging. Free elimination should be encouraged by the use of purgatives and the drinking of plenty of water. Old and cachectic individuals require careful nursing. Aspirin or the salicylates, in combination with codein, not only relieve the pain, but apparently shorten the duration of the disease.

TUMORS OF THE NERVES

Tumors of various kinds may arise from the nerves, or be localized in their region. The nerve-fibers may either pass through or be compressed by the tumor. True neuromata are very rare, most of the new-growths involving the nerves being neurofibromata, neurosarcomata, etc., which have their origin in the perineurium and epineurium.

Tumors of the nerves may be solitary or multiple. Multiple tumors may be limited to the area of one nerve, or may involve the nerves of the entire body. The size and consistence of these tumors vary greatly.

Tubercula dolorosa is a name applied to tumors, about the size of a pea or smaller, found singly or in great numbers on the sensory cutaneous nerves. They can be easily felt, and are usually very painful.

Recklinghausen's disease, general neurofibromatosis, is a condition in which numerous fibromata occur on the branches of the nerves. Pigmented patches appear on the skin, and these are sometimes accompanied by the presence of nevi and angiomata. In this condition the tumors of the nerves vary in size, and are found not only in the skin, but on the visceral nerves, especially in the mesentery peritoneum, on the sympathetic nerves, the tongue, the mucous membranes, etc.

Very little is known as to the *etiology* of this condition. Tuberculosis and injury are believed to be predisposing causes. As a rule, the tumors are of congenital origin, and often a distinct heredity or familial tendency is seen.

Symptoms.—In some cases symptoms may be absent, and the tumors discovered by accident. In other cases the tumors manifest themselves by intense pain. Usually they are very tender to pressure, and their development is accompanied by hyperesthesia, and rarely anesthesia. Paralysis and atrophy also develop. The patient's general condition is usually unaffected, unless pain is severe, when general wasting takes place.

The disease is, as a rule, a slowly progressive one, and creates a predisposition for the development of malignant tumors at other sites.

Tumors may develop on the cut end of an injured nerve. These growths may be very painful and tender. They may also occur in the neighborhood of nerves, and by their growth infiltrate or compress the nerves.

Treatment.—If tumors are single and give rise to severe pain, they should be extirpated. Tumors that develop on the proximal end of a cut nerve frequently recur after excision. The alleviation of pain is very important in these conditions, and is best accomplished by the administration of the coal-tar products, codein, morphin, etc. In some cases it is necessary to inject alcohol into the proximal portion of the nerves; in others resection of the more painful tumors is indicated. The patient's general condition should also receive careful attention.

NEURALGIA

Neuralgia is a disease characterized by the onset of pain, more or less severe in character, which occurs in attacks of varying frequency and duration. It is felt in the main trunk of a nerve or in its branches, or in both. The disease may occur at any time of life, but is very rare in childhood and more common after middle life. Puberty, pregnancy, the puerperium, and the climacteric period temporarily increase the predisposition to neuralgia.

Pathology.—The pathologic changes that take place in the nerves or ganglia are neither constant nor well marked. In some cases there is apparently a low-grade neuritic or perineuritic change, or there may be low-grade inflammatory changes in the ganglia. In other cases a sclerosis of the smaller vessels that go to the nerves or the ganglia on the nerves occurs. It is probable that the primary cause is a disturbance of the nutrition of the nerve or the ganglia, giving rise to irritation.

Etiology.—The most important predisposing cause seems to be a neuropathic disposition. In many cases there is a marked hereditary history, the disease having occurred in the family or in the ancestry. Among the commoner predisposing causes are exhausting illnesses, severe hemorrhage, anemic conditions, old age, arteriosclerosis, constipation, toxic agents, such as

alcohol, lead, mercury, tobacco, or arsenic, or it may be the result of toxins retained in the system in kidney disorders, diseases of the liver, rheumatic or gouty conditions, in diabetes, and in various metabolic disturbances. Gastro-intestinal disorders predispose to and frequently excite attacks of neuralgia, and compression or congestion of a nerve, infectious diseases, exposure to cold, physical exhaustion, or emotional excitement are all factors in the etiology. Reflex irritation, such as eye-strain, adenoids, etc.; inflammatory conditions in the neighborhood of nerves or injury to nerves or to the surrounding tissues; meteorologic changes and certain electric states of the atmosphere may predispose to neuralgia. The attacks seem to occur more frequently in winter than in summer.

Symptoms.—The chief symptom is pain, which comes on in paroxysms. Each paroxysm may last for several seconds or recur at intervals of seconds or minutes, and the entire attack may continue from a few minutes to several hours or days. The pain is described as a stabbing, boring, tearing, or burning sensation, which occurs along the distribution of a nerve. During the interval between paroxysms the patient is usually free from pain, but may experience a sensation of soreness. During the attack the nerve is generally tender, and pressure on such a nerve may bring on an attack. Sometimes the paroxysms of pain are accompanied by or immediately followed by reflex automatic spasms in the muscles, whose motor centers are in close relation to the sensory root involved.

The attack is often accompanied by secretory, motor, and vasomotor symptoms. Loss or graying of the hair, falling out of the teeth, and herpetic eruptions are among the frequent trophic disturbances that occur in neuralgic conditions. Paralysis does not occur, but the pain may give rise to restriction of active movement. The skin of the affected parts is often hyperesthetic, whereas anesthesia never develops, and decreased sensibility occurs only in the later stages.

Pressure points, *i. e.*, circumscribed spots that are tender to pressure, are usually found in the course of the affected nerve. These points generally correspond to the site at which the nerve emerges from a canal in the bone, or to a site at which it

can be pressed against solid tissue. The nerve is seldom sensitive to pressure throughout its entire course.

When the disease is of long duration, the general health of the patient is, as a rule, affected. There may be loss of appetite, digestive troubles, disturbance of nutrition, etc. In prolonged severe cases there may be marked depression, and delirium with attempts at suicide may develop.

A **diagnosis** of neuralgia should never be made before a thorough examination has excluded organic disease of the nerves or of the central nervous system, disease processes in the neighborhood of nerves, etc. The diagnosis must also be differentiated from painful attacks occurring in hysteric individuals, in whom the pain is of mental origin. This last condition is called *psychalgia*. Differentiation may be made from the fact that in the latter mental change is capable of influencing the pain, which is increased, abolished, allayed, or produced according to the idea or emotion that has occasioned it. Moreover, the pain develops or increases under observation, and disappears when the attention is diverted.

These pseudoneuralgias are also not confined strictly to nerve-tracts, nor are they constant in their distribution, frequently recurring in different areas. They are, as a rule, more diffuse in distribution, more likely to affect segments of the body, such as the entire head or half of the head, an entire arm or leg, or one-half of the body, etc. Not infrequently they are accompanied by other hysteric symptoms.

Myalgia is differentiated from neuralgia by the distribution, character of pain, and the mode of onset.

Course and Prognosis.—Neuralgia may have an acute onset, may continue a short time, and then disappear. It usually comes on in attacks that last for days, weeks, or months, with intervals of considerable duration in which there is more or less complete freedom from pain. In other cases there is simply a remission of pain, which may last for a longer or shorter time. In still other cases the attacks follow each other in rapid succession, the interval of freedom being limited to a few hours.

The **prognosis** is favorable in recent cases, occurring in young, well-nourished individuals. The neuralgias that occur with

acute onset following the infectious diseases usually respond to treatment. Those occurring in individuals with a neuropathic heredity, and in older people after long-continued, exhausting disease or chronic poisoning, present a very unfavorable prognosis.

Treatment.—Before instituting treatment a most careful and systematic examination of the patient should be made in order, if possible, to discover the cause. This should, when discovered, receive the proper treatment. In case of deficient nutrition, etc., improvement of the health by diet, exercise, etc., may effect a cure. If the exciting cause is a too rich diet, proper regulation of the food may be sufficient. In neuralgia the diet should be mixed and non-stimulating. This is especially true of diabetic and gouty individuals. Susceptibility to rheumatic influences is to be combated by proper treatment of the throat and nose and by hydrotherapy. The bowels should be regulated, and sufficient physical exercise be taken. If malaria is suspected, quinin should be administered. When syphilis is believed to be present, mercury and the iodids are to be used. In case of chronic poisoning, the source is to be removed and efforts made to eliminate the poison. For the pain, counter-irritation or blisters may not only relieve, but even cure, some cases. Ointments containing opium, belladonna, or chloroform may give relief. The local application of heat, or sometimes of cold, has a palliative effect, and in some cases electricity seems to give relief. The galvanic current is to be used, the positive pole being placed upon the affected nerve at its point of origin, or upon a pressure point, with the negative pole placed at some distance. A weak continuous current is passed through the nerve, the strength of the current being gradually increased and the current turned off and on slowly. The application should last from one to thirty minutes, and the current should never be strong enough to give rise to discomfort. Massage is of considerable benefit in some cases, especially where it is used to promote the general health. Change of climate may be necessary. Drugs are used to relieve pain and to cure the disease. Quinin in increasing doses, pushed to its physiologic limit, has proved beneficial in some cases. Arsenic and iron are

especially useful in anemic individuals. Sodium salicylate, salol, and aspirin are beneficial in rheumatic conditions. Potassium iodid is frequently of value, and aconitin and nitroglycerin are often effective where blood-pressure is high. Phenacetin, antipyrin, phenalgin, pyramidon, etc., may be given to relieve pain. It is frequently found that a drug which at first gave relief, after a time is no longer effective, and under these conditions other remedies must be tried. Later it may be found that the original drug is again effective.

Some cases are very intractable, and in these codein or morphin must be used. In obstinate cases it is advisable to inject the morphin in combination with atropin into the neighborhood of the nerve. Ointments containing analgesics may be rubbed in over a nerve, or ethyl chlorid, in the form of a spray, may be applied over the affected nerve. The subcutaneous injection, into a nerve, of 1 or 2 drops of a 1 per cent. solution of osmic acid or carbolic acid may be tried, but has no positive value. Injection into a nerve or its immediate vicinity of an 80 per cent. solution of alcohol containing a very small amount of cocain and chloroform, as advised by Schlosser and Lange, gives very satisfactory results. In those cases in which there is evidence of local irritation, surgical measures may be adopted. Those cases that do not yield promptly to medical treatment after a proper interval had better be dealt with surgically—simple section of the nerve merely gives temporary relief, and is mentioned only to be condemned. Excision of a large portion of a nerve may give permanent relief. Nerve-stretching may also have a good effect, and extraction of the nerve by means of special instruments and methods has been very successful. Intracranial or intraspinal section of sensory roots of nerves is to be recommended in intractable cases.

NEURALGIA OF THE INDIVIDUAL NERVES

Trigeminal Neuralgia; Neuralgia of the Fifth Nerve.—This is the most common form of neuralgia. It occurs in women more frequently than in men. In the majority of cases the onset

is between the twenty-fifth and fiftieth years, although no age is exempt. As the disease may be chronic in course, it may exist in individuals who are very old. There is a distinct hereditary tendency to develop this disease. In addition to the usual causes of neuralgia, disturbances of vision, eye-strain, ear diseases, diseases of the nose and its accessory sinuses, diseases of the teeth and of the bones of the face and skull, may predispose to or excite this form of neuralgia. Exposure to cold, the eating or drinking of very hot or very cold substances, emotional disturbances, and the like are particularly prone to excite an attack in individuals who are predisposed.

The principal pathologic changes observed in this condition are degeneration and low-grade inflammation of the branches of the fifth nerve, degeneration of the cells in the Gasserian ganglion, and endarteritis of the vessels of the nerve and ganglion.

Symptoms.—The chief symptom is intense pain along the course of the nerve. It occurs in paroxysms, and is of a sharp, shooting character. It may involve all the branches of the fifth nerve, or may be limited to one or more branches of the nerve. The most common form is the neuralgic pain that is limited to the supra-orbital branch, in which the pain is felt above the eye, over the forehead, sometimes in the eyeball, etc.; when the infra-orbital branch is affected, pain is felt upon the cheek, in the upper teeth, and nose; when the third or lower branch is affected, pain is felt upon the cheek and lips, in the lower teeth, and sometimes in the mouth and tongue. In addition to the pain there are frequently present disturbances of secretion, of sensation, vasomotor, trophic, and motor symptoms. During the attack there may be increased salivation, watering of the eyes, and increased nasal secretion, a flushing of the face, with injection of the conjunctiva and swelling of the eyelids; a hyperesthetic condition of the skin, numbness, or at times a decrease in sensation, and herpetic eruptions and falling out or turning gray of the hair. Motor disturbances are not uncommon in the severer forms of facial neuralgia, and when they exist, consist of convulsive movements of the muscles of the face. These occur with the paroxysm of pain (tic douloureux). During an attack the nerve-trunks are very tender at their

exit from their bony canals, at the supra-orbital notch, the infra-orbital foramen, and the mental foramen.

Course and Prognosis.—This form of neuralgia usually begins as an acute attack. It may respond promptly to treatment, and patients may never have another attack, or a second attack may occur in a few days, weeks, months, or years. This second attack may again respond to treatment, or the attacks may occur with increased frequency until the intervals of freedom are very short—the patient being free from pain only for a few days, hours, or minutes. In other cases the pain is continuous, and the only relief the patient gets is that afforded by medical or surgical treatment. The prognosis of a single attack is good, and it is only when the attacks occur with increasing frequency and are of greater severity that the prognosis is unfavorable, unless surgical intervention affords relief.

The *diagnosis* of facial neuralgia is readily made, the only difficulty being to determine the cause, and to differentiate between a true neuralgia and a symptomatic neuralgia.

Treatment.—A careful examination should be made to determine the cause and to rectify or remove this if possible. The general health of the patient must be considered, and any diseased condition should receive proper treatment. Change of climate may be desirable. Pain must be relieved, and in severe cases opium or one of its derivatives must be used. In this class of cases there is great danger of the patient acquiring the opium habit, and every effort should, therefore, be made to avoid the use of this drug. The various coal-tar derivatives, analgesic local applications, electricity, counterirritation, heat and cold, etc., should all be tried before prescribing opium. If the patient does not respond to medicinal treatment, it is unwise to wait until the patient's physical condition is run down before surgical interference is advised.

The various surgical methods of treatment are as follows: Cutting the branches of the fifth nerve at their points of exit from their bony canals—a method that is mentioned only to be condemned; the injection of the nerves at these points with osmic acid or phenol—a procedure likewise to be cautioned against; the digging and tearing of nerves from their canals—a method

that seems to give relief for a few months or longer; injecting alcohol into the nerves at their point of exit from the cranial cavity, according to the method of Schlosser and Lang—this, on account of the difficulty and unpleasant complications that may arise, should be undertaken only by one who has had the proper experience: it gives relief for a year or more and sometimes effects a permanent cure. In very severe cases the only certain method of obtaining relief is subdural resection of the sensory root of the Gasserian ganglion. This operation should be performed only by those who have had the proper training. After this operation the affected side of the face is entirely deprived of sensation, and there is great danger of causing infective and trophic disturbances of the eyes unless proper care is exercised.

Cervico-occipital Neuralgia.—This condition is characterized by pain in the neck below the occiput and the under posterior aspect of the head. It is caused by affection of the cervical plexus, especially the great occipital nerve. Spots painful to pressure are situated over the occiput, posterior to the mastoid process, etc., and the scalp is usually very tender. This condition has the same etiology as neuralgia in general, and the prognosis, course, and treatment are the same as in that condition. The attacks usually subside under treatment, but manifest a tendency to recur in some individuals.

Brachial Neuralgia.—This condition is characterized by paroxysms of pain in the distribution of one or more of the nerves of the brachial plexus. The pain may be felt chiefly in the neck, but usually affects also the arm, forearm, and hand. There are painful points where the nerves are superficial and can easily be compressed against bone. The pain is intensified by movement. Herpes is a frequent complicating symptom.

Diagnosis is made from the paroxysmal character of the pain, radiating along the nerve-trunks, the nerve-pressure points, the absence of atrophy and paralysis, disturbance of the electric reaction, and usually increased reflexes.

The *etiology*, *prognosis*, and *treatment* are the same as in neuralgia in general.

Intercostal Neuralgia.—This condition has the same etiologic

basis as neuralgia in general. It occurs more frequently in women than in men. It evidences itself by sharp shooting pain around the side of the chest, often felt chiefly in the terminal branches of the nerve, in front of the chest, or in the axillary line. The tender points are situated in front, near the sternocostal line, in the midaxillary line, and near the spinous process. Herpes is a frequent complication.

Diagnosis.—The condition must be differentiated from organic disease of the nervous system or spinal cord, from disease of the lungs, pleura, heart, etc. The diagnosis is made from the sharp, shooting character of the pain, the existence of the painful points upon the chest at the point of exit of the nerves, and by the limited distribution of the affection.

Treatment is that of neuralgia in general.

Mastodynia—Neuralgia of the Breast.—This is a form of neuralgia limited to the nerves of the mammary gland. The pain comes on in paroxysms, and may be extremely severe. It is usually associated with hyperesthesia of the skin and excessive sensibility of the nipples. Pressure-points are situated, as a rule, under the spinous process of the second to the sixth thoracic vertebræ. The disease occurs almost exclusively in middle age, and practically always in women. Anemia and a neuropathic tendency are predisposing causes. The attacks occur more frequently during pregnancy and lactation and after injury or emotional excitement. The disease is also more likely to occur in women whose breasts are pendulous.

The *diagnosis* is made by excluding tumors, organic disease of the nervous system, and hysteric states. The *prognosis* is that of neuralgia in general. The *treatment* consists in applying light compression bandages, and is that of neuralgia in general.

Lumbar and Abdominal Neuralgia.—This condition is similar to intercostal neuralgia, except that the pain attacks the lumbar and abdominal regions of the body. This type of neuralgia is also especially apt to be attended by herpes. The cause, course, prognosis, and treatment are those of neuralgia in general.

Crural Neuralgia.—In this condition the painful attacks may occur either on the exterior surface of the thigh, as far as the knee-joint, when the external cutaneous nerve is affected, or may

involve the inner surface of the thigh and leg as far as the ankle. The condition occurs more frequently in men than in women. It develops usually as the result of hard muscular work, long marches, exposure to cold, or injury. It also occurs frequently along with sciatica. Painful points are found in the inguinal canal, on the front of the thigh, on the inner side of the knee, and on the inner malleolus. Herpes is frequently an accompaniment.

Prognosis and *treatment* are those of neuralgia in general.

Sciatic Neuralgia—Sciatica.—This is a somewhat common form of neuralgia, occurring more frequently in males than in females, and usually after the twentieth year. Among the frequent causes of sciatica are exposure to cold, gouty and rheumatic conditions, and chronic intoxication of any sort. Diabetes is especially likely to be complicated by sciatic and crural neuralgia. Pressure on the nerve within the pelvis, from accumulations within the rectum, tumors, a pregnant uterus; pressure on the nerve external to the pelvis; relaxation of the sacro-iliac joint, causing either pressure or dragging of the nerve; relaxation of the plantar arches; venous congestion, etc., must all be mentioned as causes. Bilateral sciatica is particularly apt to be of diabetic origin, or symptomatic of disease of the spinal cord, spinal column, sacrum, pelvis, etc.

Symptoms.—The onset of this condition is generally indicated by a dragging sensation in the back of the thigh, in the sacral, or in the lumbar region. This sensation of discomfort becomes within a short time more severe, being replaced by stabbing, burning, or boring pain which follows the course of the sciatic nerve. This pain usually passes down the back of the thigh and leg, and may involve the foot. It comes on, as a rule, in paroxysms, but may be continuous, with remissions and exacerbations, and is especially apt to be worse at night.

Movements, as a rule, increases the pain, and the patient usually endeavors to place the leg and thigh in a position of semiflexion, to keep it as quiet as possible, and to protect the sciatic nerve from pressure. Strain, coughing, sneezing, and the like are apt to excite or increase the pain. The tender points of Valleix are: one near the posterosuperior spine of

the ilium; one over the site of the emergence of the nerve at the great sciatic notch; one at the lower margin of the gluteus maximus muscle, between the trochanter and the tuberosity of the ischium; one in the middle of the thigh on the posterior surface; one hehind the knee; one behind the head of the fibula; and one behind the external condyle of the ankle. The pain is usually deep seated, and may be increased by making extension of the leg and thigh. This point is best brought out by having the patient lie upon his back, extending the leg on the thigh, and then flexing the entire leg and thigh at the hip-joint. Pain may be produced after elevating the foot one or two feet from the bed, or it may not be felt until the thigh forms an angle of 90 degrees with the pelvis. It is also increased by making abduction against resistance, of the unaffected thigh (Bonnet's sign), and by flexion against resistance, of the unaffected thigh (Martin's sign).

The gait is usually affected. The patient limps, moving the entire limb stiffly, and there is a slight flexion of the thigh on the pelvis, the leg on the thigh, and some outward rotation of the entire leg. In the more severe cases the patient is confined to bed, and with the aid of pillows the limb is kept in the most comfortable position possible. In some cases along with the pain there are spasmodic movements and twitchings of the leg. As a rule, there are no marked sensory disturbances, but there may be paresthesia. There is no paralysis or atrophy of the muscles. Herpes is a common complication of sciatica, and may be very extensive. The tendo Achillis reflex is retained, and the reflex on the affected side is often increased.

Course and Prognosis.—The disease may be of acute onset, and attended by constitutional symptoms. Generally, however, the onset is more gradual, extending over several weeks or months, the pain becoming more severe. The condition may continue for several weeks, months, or a year, and then gradually subside, or it may become progressive and a true neuritis occur. In this condition the entire nerve-trunk is tender to pressure, there is loss of the tendo Achillis reflex, disturbances of sensation, weakness or paralysis with atrophy, may occur. Relapses are particularly apt to occur in cases of sciatica,

especially after overexertion. The prospects for recovery are best in recent rheumatic and in mild traumatic cases. The prognosis is influenced by the possibility of removing the cause and of instituting proper treatment.

Diagnosis.—Sciatica must be differentiated from sacro-iliac diseases, flat-foot, intrapelvic disorders, diseases of the hip-joint, tuberculosis of the sacrum or spinal column, diseases of the spinal cord, muscular rheumatism, locomotor ataxia, diseases of the femur, intermittent claudication, sciatic neuritis, etc. The diagnosis is generally made as the result of careful physical examination.

Treatment.—The treatment consists, first, in making thorough examination to discover the cause of the condition and, if possible, to remove it. The patient should be placed at complete rest, in a comfortable position, in which the nerve is protected. Free purgation should be instituted at once. In cases due to cold or rheumatism, warm packs and counterirritation are to be employed. Internally, salicylates or aspirin are to be given in large doses every two or three hours. All movement is to be avoided, and the patient is best kept in bed. Baking of the limb in a hot-air apparatus is beneficial. In addition to these measures, if the pain is severe, it may be relieved by codein or, if necessary, morphin. After the acute stage has passed, or in the chronic condition, in addition to the efforts to remove the cause, potassium iodid should be administered, applications of alternate heat and cold, baking, immobilization by splints, properly administered massage, counterirritation, hot or hot and cold water under pressure, the continuous galvanic current, prolonged hot sitz-baths, puncture of the nerve with needles under proper antiseptic precautions, injection, into the neighborhood of the nerve, of cold normal salt solution, etc., may all be tried. Injecting alcohol into the sciatic nerve is a method to be used only in the most severe cases, as it is likely to be followed by peroneal paralysis. Nerve-stretching may be tried, especially if there is reason to believe that the nerve is embedded in cicatricial tissue.

Coccygodynia.—This is a condition characterized by severe neuralgic pain in the region of the coccyx. It occurs much

more frequently in women than in men. The pain comes on suddenly or after overexertion, and is increased by contractions of the muscles which are inserted at the coccyx. Local tenderness is generally present, it is especially apt to occur in hysteric individuals. If the condition is one of the symptoms of the hysteric state, it should not receive local treatment, for this will only accentuate the pain. As the general condition improves this symptom will disappear. Those few cases that are truly neuralgic in character should receive the same treatment that is usually given in neuralgia.

It is very important to make a careful examination and exclude painful local disease of the pelvic bones or viscera.

DISEASES OF THE MUSCULAR SYSTEM

PROGRESSIVE MUSCULAR DYSTROPHY

Etiology.—Heredity is a prominent factor in these cases, as there is usually a history of a similar condition existing either in the family or in the ancestry. Exposure to cold, overexertion, trauma, infectious diseases, etc., have all been mentioned as among the exciting causes. The disease usually appears before the age of puberty, frequently before the sixth or seventh year.

The explanation offered by Starr, that this disease is due to an inherent defect in development of the muscles, and that the muscles, not being endowed with proper vitality, after a short time wither and die, seems to me best to account for this condition.

Pathology.—In some cases there is a simple progressive atrophy of the muscle-fibers, a simple reduction in size of the individual fibers taking place. In other cases there is a degeneration of the muscle-fibers, in which they lose their striated appearance, leaving the muscles of a smooth, yellowish tint, until finally all the muscle substance vanishes, and fatty tissue takes its place. Under some circumstances there is a deposit of fatty tissue between the muscle-sheaths, with degeneration and deposit of fat in the muscle-fibers, and not only is the space formerly occupied by the normal muscle filled in with fat, but the fatty deposit may increase the actual size of the muscle, and if this goes on to any extent, pseudohypertrophy of the muscle will occur. Along with the atrophy we find occasionally true hypertrophy of individual muscle-fibers. With the

changes in the muscle tissue there is usually a proliferation of connective tissue, and if this, after a time, contracts longitudinally, there will be a shortening of the atrophied muscles.

Symptoms.—Erb, who has gathered and analyzed many facts in regard to this affection, divides the disease into two groups: (1) Those in which there is an increased size of the affected muscles—either a pseudohypertrophy or a true hypertrophy of the muscle-fibers; (2) those in which there is no increase in size of the muscles. From a clinical standpoint the following types of the disease are recognized: (1) Pseudomuscular hypertrophy of Duchenne; (2) Erb's juvenile dystrophy; (3) Duchenne-Landouzy-Déjérine type of dystrophy.

The first type, or *pseudomuscular hypertrophy of Duchenne*, makes its appearance in earliest childhood. It affects males more frequently than females. It involves by preference the muscles of the calf, thigh, pelvis, and lumbar region. The increase in the size of the muscle extends over large areas, and is especially apt to appear in the lower extremities. The second type, or *Erb's juvenile dystrophy*, is more apt to occur in youth and young adult life. It generally first involves the muscles of the shoulder girdle and upper arm. The third type, or *Duchenne-Landouzy-Déjérine* form of dystrophy, is characterized by primary involvement of the facial muscles. In addition to these three types many others have been described. The difference in classification arises from the fact that cases occur in which the early symptoms affect regions of the body differing from those of the three types described. The course may vary, but eventually, as the disease process progresses, all the types present a similar appearance. Although, as a rule, there is a distinct hereditary history in these cases, yet many isolated examples of the disease occur. The symptoms generally develop slowly, and often pass unnoticed for a long time.

In those cases in which the muscles of the pelvis, thigh, and spinal column are first affected, the disorder first becomes apparent in walking and in raising the trunk The gait is waddling, and difficulty is experienced in going up steps. The patient falls readily. In raising himself from the sitting position he makes use of the arms, leans forward, places his hands upon

his thighs, and in this way brings the body into the erect posture (Fig. 106). When the patient is in the recumbent position, the manner of rising is characteristic: he turns first on his abdomen, then, resting his hands upon the ground, he raises the trunk until he attains the kneeling position; then he lifts the knees from the ground, and places one hand upon the knee of the same side and the other upon the other knee; he then works

Fig. 106.—Illustrating the position patients assume in arising from a stooping to an erect position.

his hands up his legs until he attains the erect position. As the disease advances he cannot raise himself from the ground without help, or without supporting himself on some firm object. This difficulty is due to weakness of the muscles which extend the legs on the thighs, the thighs on the pelvis, etc.

Lordosis of the lumbar spinal column is also usually a prominent symptom. It is caused by weakness of the muscles that keep the pelvis extended on the thighs, allowing an abnormal

inclination of the pelvis forward and downward to take place. When the shoulder group of muscles is involved, the shoulder-blade becomes abnormally mobile, there is a drooping of the shoulders, and the shoulder-blades stand away from the throax. There is marked interference with the movements of the shoulder

Fig. 107.—Patient showing lordosis in the erect position, which disappears when he is seated.

and arms (Fig. 107). In those cases in which the muscles of the face are involved there is frequently pseudohypertrophy of the lips, the mouth is held slightly open, and the patient is unable to whistle or to pout the lips. There is frequently an inability to close the eyelids completely (Fig. 108).

The affection may commence in any part of the body, but the three types that have been described are characteristic of the more common forms of onset.

The muscles most frequently affected are the trapezius, the serratus magnus, latissimus dorsi, infraspinatus, deltoid, biceps, supinator longus, erector spinæ, pelvic muscles, the glutei, quadriceps extensor, adductors of the thighs, calf muscles and peroneal

Fig. 108.—Duchenne-Landouzy-Déjérine type of muscular dystrophy.

muscles, orbicularis oris, orbicularis palpebrarum, etc. As a rule, the proximal parts of the extremities are more markedly affected than are the distal portions. Some of the muscles involved show a decrease in size; others may show an apparent increase in size, or the atrophy or hypertrophy may be limited to a part of the affected muscles.

The deep reflexes are usually decreased and eventually lost.

The mechanical excitability of the muscles is also decreased and finally lost. The electric reaction is generally normal, and is present in proportion to the amount of muscle tissue that remains intact. There are no sensory disturbances, and fibrillary tremors are absent.

The *course* of the disease is usually protracted. The onset is very gradual, with a slow increase of symptoms. Contractures occasionally appear in certain muscles.

The *prognosis* as to life is good, the disease extending over many years. The patient usually dies of an intercurrent affection. In some cases the disease advances to a certain stage and then becomes stationary. The condition may be associated with hysteria, mental weakness, epilepsy, and congenital anomalies of the bony structures of the body.

Diagnosis.—The peculiarity of the figure and attitude, the coexistence of atrophy and hypertrophy, present so characteristic an appearance that there is usually no difficulty in making the diagnosis. The absence of sensory disturbances and of fibrillary tremors; the fact that the electric reaction is normal in type but present only in proportion to the amount of muscle tissue present; the age of onset; the location of the muscles involved, etc.—are all points that assist in differentiating this disease from other conditions that are characterized by atrophy and weakness, such as neuritis, the spinal form of progressive muscular atrophy, Friedreich's disease, etc. The greatest difficulty lies in recognizing the affection in its earliest incipiency, and here a careful study of the case is necessary.

Treatment.—This consists of rest, with gentle systematic exercise; general massage, properly given; electric treatment, especially the galvanic current; nourishing food, fresh air, and pleasant surroundings; and, when necessary, supporting apparatus to prevent deformity or to enable the patient to walk, etc.

POLYMYOSITIS

Polymyositis is a condition characterized by pain, tenderness, and weakness or paralysis of the muscles, the last of which comes on rapidly.

Etiology.—This condition may be acute or chronic. It affects any age, men being more prone to be affected than women. It may occur in tubercular individuals or during the puerperium, it may follow influenza, tonsillitis, gastro-intestinal intoxications, exposure to excessive cold, infection, etc.

The pathologic changes that take place are: A parenchymatous or interstitial myositis, involving all or any of the muscles of the body; serous infiltration, with a mottled, grayish-yellow appearance of the muscle tissue, which may be firm or soft in consistency.

Symptoms.—The disease is usually of acute, but not of sudden, onset. It manifests itself, as a rule, with general constitutional symptoms. There is a feeling of exhaustion, with heaviness of the limbs; this may be accompanied by headache, vertigo, and gastro-intestinal symptoms. From the first, or within a few days, there is a marked dragging pain in the muscles, and the patient gradually becomes weaker until finally he is more or less completely paralyzed. The proximal parts of the extremities are usually more severely affected than the distal parts. The muscles are tender to pressure and passive movements cause pain. The muscles are generally swollen, and the skin and subcutaneous tissues are frequently edematous and infiltrated, this being more marked over the affected muscles. The arms are, as a rule, more severely affected than the legs. The skin may be reddened, and this sometimes suggests erysipelas. Hyperidrosis is frequently present. This condition may be limited to a very few muscles, to the muscles of one extremity, or may be irregular in distribution. The muscles of respiration and deglutition, the tongue, the diaphragm, the heart, etc., are rarely affected. There are no sensory disturbances. The mind remains clear except in so far as the activity of the brain is interfered with by the action of fever, weakness, and general intoxication. The tendon-reflexes corresponding to the affected muscles are usually decreased or lost, and there is commonly a rise of temperature throughout the course of the disease. This condition must be differentiated from trichinosis, from which it is readily distinguished by the fact that the latter may affect a number of individuals who have eaten meat from the same

animal, and by the marked prominence of the gastro-intestinal symptoms at the onset of the disease. Moreover, in trichinosis the edema of the face and eyelids develops early, and there is marked increase of the eosinophile cells in the blood.

Course.—The disease may last for weeks, months, or even for several years. It may terminate in recovery in a few weeks or end in death. It may also run an intermittent course. In chronic cases remissions are not uncommon.

The **prognosis** as to life is grave, except in those cases that have assumed a mild course from the first.

Treatment consists principally of free elimination, good nursing, proper food, and hydrotherapy.

ISCHEMIC PARALYSIS (VOLKMANN'S PARALYSIS)

This condition usually occurs after an injury, such as a fracture, in which a bandage has been applied too tightly or in which, after the application of the bandage or of a plaster cast, swelling occurs. As a result of pressure there is an extensive interference with the circulation in the affected part, and the distal portion of the affected limb presents a bluish appearance and may be cold to the touch. There is usually very great pain; if the pressure is not relieved, paralysis follows, with disintegration of muscle tissue, which is followed by marked atrophy and secondary contraction.

The **prognosis** in this condition is grave, as recovery does not take place unless the condition is discovered very early.

Treatment consists in avoiding tight bandages. When pain is complained of, the bandages should always be promptly removed. If the condition is discovered early, warm applications and gentle massage are indicated. If the damage has already been done, the best that can be hoped for is relief or rectification of deformity; this is accomplished by massage, hot applications, and lengthening of the tendons.

THOMSEN'S DISEASE (MYOTONIA CONGENITA)

This condition is characterized by its marked hereditary tendency, great hypertrophy of muscle-fibers, and a proliferation of the muscular nuclei. The disease was first described by Dr. Thomsen, in whose family over twenty cases occurred in four generations. The condition generally becomes evident early in life, but may not develop until early adult life, or it may follow some mental excitment. Cold or emotional disturbances increase the severity of the affection, whereas active exercise tends to diminish its severity.

Symptoms.—The characteristic symptom of this affection is a peculiar rigidity of the muscles occurring after they have been at rest for some time. The patient finds that, after resting, he cannot voluntarily perform any act until he has made several efforts to do so; with repeated effort free movement is finally brought about, but after another period of rest the rigidity returns. In some cases the muscular stiffness becomes evident only during very cold weather. Mechanical excitability of the muscles is increased; pressure or percussion of the affected muscles gives rise to slow, tonic, persistent contrations. Upon application of the electric current the myotonic reaction is elicited. There are no disturbances of sensation or of the organic reflexes. The knee-jerk is often decreased and sometimes lost. Cases vary in severity, the very mild ones manifesting no marked symptoms.

The **diagnosis** is readily made from the hereditary character of the disease and the peculiar reaction to the electric current.

The **prognosis** as to life is favorable, but as to recovery or improvement it is very bad. There is no known treatment.

OPPENHEIM'S DISEASE (MYATONIA CONGENITA)

This is a very rare condition, occurring in early childhood, and characterized by atony of the muscles (Figs. 109, 110). It is probably due to delayed development of the musular system. The muscles of the eyes, tongue, throat, and respiration do not seem to be involved. From the very first there is noticed an

absence of the normal movements of the lower, and sometimes of the upper, extremities. There is a striking flaccidity of the affected muscles, and the tendon reflexes are weakened or lost. The muscles are not visibly wasted, but their electric excitability is diminished or lost. Active movements are feebly executed, and the child may appear to be paralyzed, yet on close examination it will be seen that a certain amount of movement is evident.

Fig. 109.—Myatonia congenita, showing inability of patient to sit erect.

The joints are usually movable, and the limbs may be placed in very abnormal positions. There are no disturbances of sensation, and at the proper age there is control of the rectal and vesical reflexes.

The **prognosis** is not good, as the majority of cases die from an intercurrent affection. In those who have survived, slight improvement has been known to take place.

Fig. 110.—Myatonia congenita, showing abnormal mobility of joints.

PERIODIC PARALYSIS

This disease is characterized by a rapidly developing general paralysis of all the muscles excepting those of respiration and deglutition. This paralysis may last for a few hours, for several days, or even longer. The condition develops within the course of a few hours, usually attacking an individual who is apparently in good health. It is not attended by constitutional symptoms. There is generally a history of similar attacks in the family or in preceding generations. This condition is

believed to be due to the action of a toxic substance upon the motor filaments of the nerves or upon the muscles.

Symptoms.—There is a comparatively rapid loss of muscular power, unattended by pain or sensory disturbances. The paralysis is flaccid in type, and there is a loss of the tendon-reflexes, with complete loss of muscle contraction to the galvanic or the faradic current. The paralysis may be very extensive, involving all the muscles, or it may be limited to some of the muscles of the upper and lower extremities. The attack may come gradually over a period of several hours and continue for hours or days, the patient gradually recovering strength and returning to the normal. The seizures usually occur in paroxysms, which may extend over a period of several weeks. The individual paroxysm may be mild or severe, a mild one frequently being followed by a severe one. As a rule, there is an interval of several months or years between attacks.

Treatment.—As the causative agent has not been discovered, treatment is, to a large extent, symptomatic. It consists of elimination by the use of purgatives, diuretics, etc. Alkalis seem to have a beneficial effect. Atropin is indicated if there is any respiratory embarrassment, and strychnin in proper doses should be given throughout the attack.

DISEASES OF THE CRANIAL NERVES

DISEASES OF THE FIRST OR OLFACTORY NERVE

Etiology.—This nerve may be affected in acute or catarrhal conditions of the nasal mucous membrane, disturbances of the nasal secretion, tumors in the nose, diseases of the ethmoid bones or ethmoid cells, diseases within the anterior fossa of the skull which bring about compression or involvement of the olfactory bulbs or tracts, such as basilar meningitis, fracture of the skull, tumors of the brain, etc.

Symptoms.—If only one of the olfactory nerves or tracts is destroyed, there is usually no evidence of the condition. In those cases in which the conductivity of both nerves or tracts is interfered with there is loss of the sense of smell or *anosmia.*

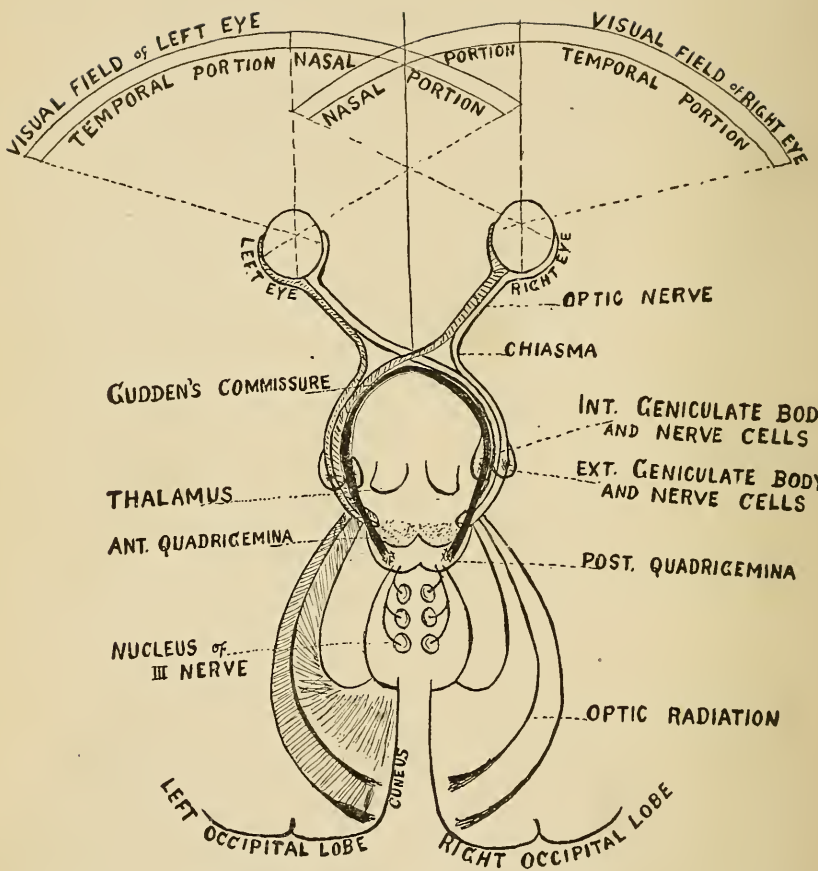


Fig. 110 *a*.—Showing course of visual impulses from point of apparent origin in the visual fields to the centers of visual perception (diagrammatic).

Irritation of one or both tracts may give rise to a condition in which the patient perceives an odor, either agreeable or disagreeable, without the true existence of an odor; this is a true hallucination of smell, and is called *parosmia*. Disturbance of the function of the first nerve is of interest principally from the assistance it gives in diagnosing diseases of the structures with which it comes into apposition. Primary disease of the first nerve itself is extremely rare. The treatment of the condition is the discovery of the cause and its removal, if possible.

DISORDERS OF THE SECOND OR OPTIC NERVE

Before taking up a study of the diseases of the optic nerve, it is well to refer to the diagram. (See Fig. 110*a*.) This diagram demonstrates that symptoms arising from involvement of the various portions of the visual pathway vary widely. When the terminal filaments of one optic nerve are affected, or when one optic nerve is diseased, there is disturbance of vision in the eye supplied by that nerve. When an optic tract or pathway posterior to the tract is affected, there is disturbance of vision in one-half of each eye, a condition known as *homonymous hemianopsia*. In the optic chiasm the symptoms depend upon the extent of involvement and the position of the lesion. If the lesion is very extensive, there may be partial or total blindness. If the lesion involves only the lateral aspect of the chiasm, the fibers that originate in the temporal portion of one eye are affected, and the condition gives rise to blindness in the inner portion of the visual field of one eye, *i. e.*, *unilateral nasal hemianopsia*. If the lesion involves only the anterior or the posterior part of the chiasm, the fibers that come from the nasal half of both retinæ are involved; this gives rise to blindness in the outer portion of the visual fields of both eyes—*bilateral temporal hemianopsia*. Central vision is usually affected only in diseases of the nerve itself. Examination for disturbance of vision is made by testing the acuteness of vision and determining the visual fields for both eyes and for each eye. In addition to the stimuli that result in vision, other stimuli are transmitted by the optic nerve which reflexly affect the nuclei of the third nerve, and, through this nerve, result in contraction and dilatation of

the pupils. Diseases that prevent the transmission of the stimuli through the optic nerve or from the optic nerve to the third nerve, and through these to the ciliary muscles, may bring about disturbance of the pupillary action. For the more technical details of examination, etc., the reader is referred to the various text-books on ophthalmology. For the general methods of examining for visual and pupillary disturbances reference should be made to the chapter on Examination of the Patients.

Edema of the Optic Nerve, or Choked Disk.—In this condition there is an elevation of the optic disk, attended by venous

Fig. 111.—Choked disk.

congestion. This may take place without impairment of the vision. There is usually no evidence of inflammation, but small hemorrhages into the retinæ may occur. This condition is generally secondary to increased intracranial pressure, or may arise in any condition in which there is obstruction to the circulation in the eye. It is frequently present in cerebral tumors, abscesses of the brain, extensive hemorrhages into the brain, meningitis, edema of the brain, etc. If choked disk continues for any length of time, the vision is usually impaired. In some cases visual disturbances occur early. The only pos-

itive means of making the diagnosis is from an ophthalmoscopic examination. Treatment consists in removing the cause, and if the latter connot be rapidly discovered, decompression operations are justifiable (Fig. 111).

Optic Neuritis.—This condition is usually secondary to other conditions, and may be caused by exogenous toxins, such as alcohol, tobacco, or arsenic; or endogenous toxins, as in nephritis, diabetes, or gout; or it may occur during the course of infectious diseases, in severe anemia, syphilis, after exposure, in meningitis, etc. In addition to the secondary forms of optic neuritis, there

Fig. 112.—Neuroretinitis.

also occur occasionally cases in which no cause is discoverable. Optic neuritis may be a hereditary condition, in which case the symptoms usually occur in young adult life and progress until blindness ensues (Fig. 112).

Optic neuritis is generally bilateral, although one nerve may be affected before the other, and one may show more extensive involvement than the other.

Symptoms.—These consist of sensitiveness to light; objects appear indistinct, and there seems to be a haze or cloud before the eyes, or, less often, rapid loss of vision, and finally, unless the condition is arrested, blindness ensues.

Diagnosis is made as the result of ophthalmoscopic examination. The *prognosis* depends upon the possibility of removing the cause. If the cause can be removed before the nerve has been completely destroyed, partial vision is regained.

Treatment consists in removing the cause, if possible. Complete rest of the eyes should be enjoined. In some cases cerebral decompression is indicated. Counterirritation over the temples is also useful. Potassium iodid in proper doses and, after the acuteness of the disease has passed, strychnin should be given.

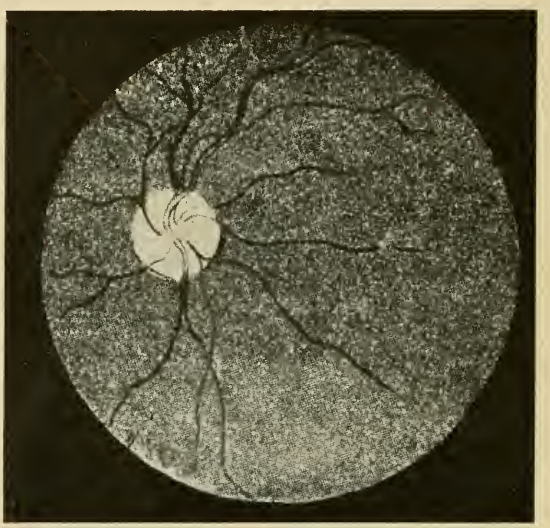


Fig. 113.—Simple atrophy of the optic nerve.

Optic Atrophy.—This condition may occur as a primary affection, or it may be secondary to some other disease. As a primary affection it may be congenital in origin or appear shortly after birth, when it is an evidence of neural insufficiency. It may occur without any preceding disturbance of the optic nerves, as in such diseases as locomotor ataxia, multiple sclerosis, diabetes, and chronic alcohol and tobacco poisoning (Fig. 113). As a secondary affection it may be the terminal state of an optic neuritis or of other diseased conditions of the optic nerve (Fig. 114).

Symptoms.—Optic atrophy evidences itself by a gradual loss

of vision. This is accompanied by a progressive contraction of the visual field and defective color vision.

Diagnosis is made as the result of ophthalmoscopic examination, inspection of the fields of vision for white and for colors, etc. In optic atrophy it is important to discover the underlying cause, if one exists. The *prognosis* is very grave, and unless the cause is removed the condition is progressive.

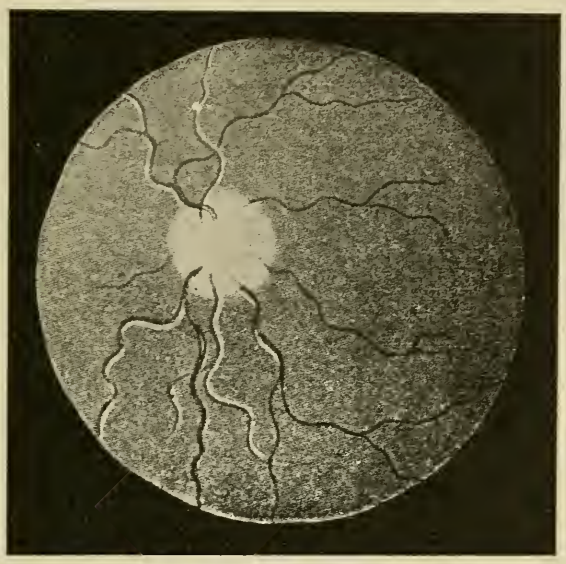


Fig. 114.—Postneuritic optic atrophy.

Treatment.—Remove the cause, build up the general health of the patient, and give strychnin in increasing doses to the physiologic limit.

DISEASES OF THE MOTOR NERVES OF THE EYE

Diseases of the Third Nerve.—This nerve may be affected in diseases involving the base of the brain in the middle fossa, such as meningitis, tumors, abscess, hemorrhage, syphilis, etc. It is also affected in diseases within the orbit, such as fractures, tumors, etc., and may be involved as the result of infectious diseases or be one of the evidences of chronic toxemia. Exposure, thrombosis of the cavernous sinus, chronic alcoholism, toxemia caused by eating infected fish, meat, or sausage, arteriosclerosis

of the basal cranial vessels, etc., are all predisposing causes. Syphilis is one of the most common causes of paralysis of the third nerve.

Symptoms.—As this nerve supplies the levator palpebræ, internal rectus, superior rectus, inferior rectus, and sends branches to the ciliary muscle, the symptoms are dependent upon the location of the lesion affecting this nerve. If the nerve-stem is involved between its point of origin and its entrance into the orbit, before dividing into its separate branches, this is evidenced either by weakness or by paralysis of all the muscles supplied by this nerve. In this condition there will exist a ptosis or drooping of the upper eyelid, inability to turn the eye-ball upward, downward, or inward, with loss of the pupillary reflex to light and to accommodation. If the third nerve is affected within the orbit, one or more of the branches may escape, and the symptoms will then be limited to the particular branches involved. If the paralysis of the levator palpebræ is complete, so that the patient is unable to raise the upper eyelid sufficiently to see, he usually makes an extra effort to elevate the upper eyelid by elevating the eyebrow, thus causing excessive wrinkling of the skin of the forehead.

The *prognosis* depends entirely upon the cause. If the condition is due to syphilis, exposure to cold, or occurs after infectious diseases or toxemias, the prognosis is usually good.

Treatment.—This consists in treating the causative condition and in administering strychnin in proper doses.

Paralysis of the Fourth Nerve.—The fourth nerve supplies the superior oblique muscle. Isolated paralysis of this nerve is extremely rare. The nerve is frequently affected along with the third nerve, it being commonly involved in the same conditions that affect the latter nerve. When the fourth nerve is affected, there is difficulty in rotating the eye upward and outward.

Paralysis of the Sixth Nerve.—The sixth nerve supplies the external rectus muscle. It pursues the longest course within the cranial cavity of any of the cranial nerves, and is frequently affected. The same causes that bring about involvement of the third or fourth nerve may also affect the sixth nerve.

Symptoms.—Involvement of the nerve brings about weakness

or paralysis of the external rectus muscle, and this results in either partial or total inability to rotate the eyeball outward; as a result, double vision frequently occurs.

Ophthalmoplegia.—This is a condition characterized by partial or complete paralysis of the muscles of the eye. It may result from involvement of the peripheral portions of the third, fourth, and sixth nerve, or from involvement of the nuclei or of the nerves in their course from the nuclei to their superficial origin.

Etiology.—The disease may be caused by exposure to cold, or may follow or accompany infectious diseases, diphtheria, influenza, typhoid fever, syphilis, injury, trauma, inflammatory conditions at the base of the brain, chronic intoxication, compression by tumors, exudates, or hemorrhage, disease of the blood-vessels, etc. It may also occur as the result of a lesion involving the brain-stem.

Symptoms.—It may come on gradually or suddenly. Paralysis may be complete or partial, some or all of the muscles of the eye being involved, or only the internal muscles of the eye may be affected, when it is known as *internal ophthalmoplegia.* When the external muscles of the eye alone are involved, the affection is known as *external ophthalmoplegia.* It may be bilateral or unilateral, and may occur as a part of some more diffuse process. The condition is manifested by partial or complete immobility of the eyeball and upper eyelid in external ophthalmoplegia, and partial or complete immobility of the pupil in internal ophthalmoplegia. When it is due to an involvement of the nuclei, the symptoms are usually bilateral; when due to involvement of the peripheral portions of the third, fourth, and sixth nerve, the symptoms are dependent upon the location, extensiveness, and character of the causative condition. Acute ophthalmoplegia is, as a rule, sudden in onset, with severe constitutional symptoms.

The *course* of the disease depends upon the causative condition; if this is hemorrhage or infection, there is usually a rapidly fatal termination; if due to thrombosis or edema, there is generally some improvement at first, but permanent symptoms of irregular ocular paralysis remain. The disease may be chronic

in onset, beginning gradually, one muscle after another being affected. In these cases the lesion is generally vascular, the result of a tumor or syphilis.

The *prognosis* depends upon the cause: those cases due to exposure, occurring after toxemias, infectious diseases, the result of syphilis or vascular diseases, if not very extensive, usually recover; in chronic extensive cases the prognosis is very unfavorable.

Treatment.—This should be directed toward removing the cause. In cases of acute onset, with fever, ice-bags should be applied to the back of the neck and over the temples, and blood-letting in the temporal region and free purgation should be immediately instituted. In the chronic conditions potassium iodid and strychnin, in addition to the treatment of the cause, may be effective.

DISEASES OF THE FIFTH NERVE

With the exception of neuralgia, primary isolated diseases of the fifth nerve are rare. This nerve supplies sensation to the face, to the mucous membrane of the eyes, nose, and mouth, and, through its chorda tympani branch, it supplies the sense of taste to the anterior two-thirds of the tongue.

Etiology.—This nerve may be affected in morbid processes developing at the base of the brain, from the pons and meninges, such as fractures, inflammation, and new-growths; in diseases of the sinuses, syphilis, and operations for the removal of the Gasserian ganglion; it may be involved in infectious diseases and toxemias. In tetanus especially there is a selective tendency toward involvement of this nerve. Hemorrhage, softening, tumors, etc., involving the pons also bring about disturbances in the functions of the fifth nerve.

Symptoms.—The symptoms pointing toward involvement of this nerve are of two kinds: those due to irritation and those due to destructive processes. As this nerve is a mixed one, containing both sensory and motor filaments, irritative conditions may manifest themselves along the distribution of either the sensory branches or the motor branches, or both. The evidences of irritation of the sensory branches are pain or paresthesia in

the forehead, eye, face, nose, mouth, and teeth, depending upon the extent of the involvement. Irritation of the motor portion evidences itself in spasmodic contractions of the muscles of mastication. Destructive lesions of this nerve may manifest themselves by loss of sensation in the distribution of one or more of the branches of the nerve, with or without paralysis of the muscles of mastication. In addition to the motor and sensory functions, the fifth nerve also transmits vasomotor and trophic stimuli. In destructive diseases of this nerve it is quite common to see ulcerations, disturbance of the nutrition of the teeth and gums, disorders of the secretions of the mucous membrane of the nose and mouth, herpetic eruptions, loss of the winking reflex, and disturbances in the reflex secretion of tears, saliva, etc.

Prognosis and **course** are dependent upon the causative condition. The treatment should be directed toward removal of the cause.

DISEASES OF THE SEVENTH OR FACIAL NERVE

Etiology.—In about 70 per cent. of cases of facial paralysis occurring in adults, in which the peripheral portion of the nerve is affected, exposure to cold is the cause. This rheumatic affection of the nerve is frequently combined with a congenital smallness of the stylomastoid foramen. Disease of the middle ear, with necrosis of the petrous portions of the temporal bones, is an extremely frequent cause in infancy and childhood. The condition may also follow diphtheria, mumps, erysipelas, gout, or diabetes, and may occur during the puerperal period, in injuries, such as the application of forceps during delivery, fracture of the skull, operations upon the mastoid, chronic intoxications, syphilis, inflammatory diseases, new-growths, and vascular diseases at the base of the brain.

Diseases of the pons, medulla, internal capsule, or lower portion of the precentral convolution may also cause weakness or paralysis of the facial muscles. Facial paralysis may be congenital, in which case it is usually bilateral, and associated with paralysis of other cranial nerves or other anomalies of development.

Symptoms.—The symptoms of disease of the seventh nerve are weakness and paralysis of the muscles of expression. The type of paralysis may be flaccid or spastic. The extent of the paralysis depends upon the severity and position of the lesion, and whether the nerve is affected externally to the stylomastoid foramen, within the Fallopian canal, in its course between the medulla and internal auditory meatus, where it is in apposition with the eighth nerve; in the medulla, where it is in close relationship with the nucleus of the sixth; or whether the disease is due to involvement of the lower precentral convolution or the tracts that convey the impulse from this convolution to the nucleus (Figs. 33, 73). On the nerve, just before entering the internal auditory foramen, is a small ganglion—the geniculate ganglion. When this is involved along with the nerve, there is usually pain in the ear and mastoid region, and disturbance of sensation in the external auditory meatus, and frequently a herpetic eruption over the tympanum and external auditory canal occur. In those cases in which the disease involves the nerve close to the stylomastoid foramen the onset is more or less acute, depending upon the cause. When the condition follows chill or exposure it is called *Bell's paralysis* (Fig. 115). In this form a day or so after exposure the patient, upon arising, notices that there is a difference in the appearance of the two sides of the face. There is little or no pain, but there may be the general constitutional symptoms as in a cold. Depending upon the extent of involvement of the nerve, the patient will notice either a total or a partial paralysis of the muscles of the face. Thus on one side there may be a flatness, the natural lines of the affected side of the face being obliterated. There is an inability to wrinkle the forehead and to close the eyelids; the reflex act of winking is abolished. The patient is unable to show the teeth, to whistle, or to perform other voluntary movements with the affected side of the face. In chewing, the food collects between the cheek and the gums. The appearance of the face is very characteristic, and, owing to the absence of the reflex movements of the eyelid, there frequently occurs a conjunctivitis and tears run from the corner of the eye. In these cases the paralysis is of a flaccid type, and according to the severity of the condition do we have com-

plete or partial reaction of degeneration. When the nerve is affected within the Fallopian canal, the corda tympani, which accompanies it in this part of its course, is affected, and, in addition to the foregoing symptoms, there is disturbance of the sense of taste over the anterior two-thirds of the tongue on the affected side. When the nerve is affected just internal to the internal auditory meatus the geniculate ganglion may be involved, in which case there is pain in the ear, sensory disturbance in the external auditory canal, and, most probably, herpetic eruptions, in addition to the paralytic symptoms. When the nerve is affected in its course from the medulla to the internal

Fig. 115.—Bell's palsy.

auditory meatus, owing to the fact of its close apposition to the eighth nerve, there are nearly always auditory disturbances added to the paralytic symptoms. If the nerve, in its course from the nucleus to its superficial origin, or if the nucleus itself is involved, owing to the close anatomic relationship of the nucleus of the sixth to the nucleus of the seventh nerve, and to the course of fibers of the seventh from its nucleus to its superficial origin, there is, in addition to the paralytic symptoms referable to the seventh, disturbance of the function of the sixth nerve.

If the facial paralysis is due to a lesion involving either the precentral convolution or the tracts from this convolution to the nucleus of the seventh nerve, the facial paralysis is never com-

plete—the forehead and the eyelids escape. The paralysis is of the spastic type, and there are no disturbances of the electric reaction. Rapidity of onset depends in large part upon the cause, some cases being comparatively sudden and acute, whereas others are very much more chronic. Bilateral paralysis is comparatively rare, but may occur as a congenital condition, as one of the manifestations of a chronic toxemia, or as an evidence of disease of the medulla. It may occur in rheumatic cases, in which one side of the face is affected after the other. In some instances but a brief interval elapses before the two sides are affected, and in others there is an indefinite period, the other side of the face being affected in the individual's second attack.

The **course** of this disease is dependent upon the location and severity of the lesion. In those cases that are due to supranuclear diseases, disease of the nucleus or of the nerve within the cranial cavity or the Fallopian canal, the progress of the affection depends altogether upon the cause, some cases being acute, whereas others are chronic. In those cases due to disease of the nerve external to the Fallopian canal the onset is usually acute, the condition coming on within a few hours or days, reaching its greatest intensity very rapidly, and then remaining stationary or gradually improving until recovery takes place.

Prognosis is dependent entirely upon the cause. In those cases in which the nerve is affected external to the stylomastoid foramen, the prognosis is usually good; in those due to diseases of the nucleus or of the trunk of the nerve within the cranial cavity or within the Fallopian canal, the prognosis is unfavorable. The electric examination of the muscles and nerve on the affected side, made about a week after the occurrence of the affection, is of the greatest assistance in making a prognosis. If the cause can be removed and the patient has normal electric reaction, recovery will take place within six weeks; if the cause can be removed and the patient has a partial reaction of degeneration, recovery will take place within six months; if the cause can be removed and the patient has a complete reaction of degeneration, the probabilities are that complete recovery will take place in from nine to eighteen months. Following recovery there

develop in some cases contractures of the affected muscles, which cause the face to appear as if there was a paralysis on the opposite side.

Treatment.—If possible, remove the cause. In those cases in which the condition is due to exposure, if the patient is seen within one or two days after the occurrence of the paralysis, it is well to apply a blister over the lower mastoid region. Internally, salicylates or aspirin in proper doses are indicated. Promote free elimination by the skin, kidneys, and intestines; keep the patient warm, and avoid all exposure. This treatment should be persisted in for about a week, after which measures should be taken to promote regeneration of the nerve, to maintain the nourishment of the muscles, and to prevent the development of unpleasant complications. Internally, strychnin in proper doses and small doses of potassium iodid or ferrous iodid should now be given. Electricity should be used daily, using that form of current which will bring about muscle contraction. Massage, properly applied, is also very useful. As the patient improves electric treatment and massage may be given less frequently. Proper protection of the conjunctiva of the affected eye will prevent the development of disagreeable complications. Contractions, if they develop, may be relieved by warm applications and massage. In those cases that show no tendency to recover after eighteen or twenty-four months surgical treatment may be resorted to.

DISORDERS OF THE EIGHTH OR AUDITORY NERVE

The eighth nerve is a sensory nerve and has two distinct functions: it transmits impulses that result in hearing, and it transmits impulses that are concerned with equilibrium.

Etiology.—The nerve may be affected in such diseases as locomotor ataxia, multiple sclerosis, etc.; in tumors, in inflammatory conditions, in diseases of the blood-vessels and bones, and in injuries at the base of the brain; in syphilis, in marked anemic conditions, following exposure, in chronic intoxications, and in disease of the middle ear or labyrinth, etc.

Symptoms.—The symptoms of disturbance of function of this nerve or its branches are defective hearing, unstable equi-

librium, vertigo, and tinnitus aurium. There may be complete or partial deafness, caused either by diseases of the nerve itself or by diseases of the ear.

Tinnitus aurium is a condition characterized by subjective sounds that apparently occur within the ear, as ringing, roaring, throbbing, etc. These sounds are due to irritation of the auditory nerve or auditory organs, and may be due to disease of the blood-vessels, disturbance of circulation, anemic or gouty conditions, diseases of the middle and external ear, conditions that interfere with the circulation of air in the external auditory canal, drugs, especially quinin, diseases of the throat and nose, general malnutrition, hysteric and neurasthenic states, long-continued subjection to loud noises, etc.

The disturbance of equilibrium and the vertigo may be due to diseases of the eighth nerve or of the semicircular canal. In addition to disorders of the eighth nerve or of its end-organs, there are many other conditions in which disturbance of equilibrium and vertigo occur, but these are not, as a rule, attended by derangements of hearing.

Prognosis.—The prognosis of disease of the eighth nerve depends entirely upon the cause.

Diagnosis.—Isolated involvement of the eighth nerve is rare compared with the frequency of ear diseases. Careful examination usually enables one to distinguish between these conditions. The occurrence of symptoms indicating involvement of other structures of the body or of other nerves enables one to locate the site of the disease. Toxic and infectious acoustic neuritis is characterized by deafness, which is generally bilateral, coming on with loud and usually constant tinnitus aurium.

Treatment.—This consists in treating and removing the cause.

DISEASES OF THE NINTH OR GLOSSOPHARYNGEAL NERVE

This nerve supplies sensation to the pharynx and the tympanic cavity, and the sense of taste to the posterior part of the tongue. It also contains some motor filaments which, in common with those of the pneumogastric, arise from the nucleus ambiguus.

Etiology.—The ninth nerve may be affected in syphilitic conditions, in inflammatory processes, tumors, fractures affecting the posterior fossa, disease of the jugular vein, tumors and injuries of the neck, diseases of the medulla, etc. It may also be involved as the result of infectious diseases, such as diphtheria, and in chronic intoxication.

Symptoms.—Isolated paralysis of this nerve is practically never observed. When the nerve is affected along with other nerves, the symptoms consist of anesthesia in the upper part of the pharynx, loss of taste in the posterior part of the tongue, difficulty in swallowing, regurgitation of food through the nose, loss of the pharyngeal reflex, and paralysis of some of the muscles of the palate.

Prognosis and **treatment** are those of the causative condition.

THE TENTH, PNEUMOGASTRIC, OR VAGUS NERVE

This nerve has both sensory and motor fibers. Its motor fibers arise from the nucleus ambiguus, in common with those of the ninth nerve. This nerve is also closely connected with the sympathetic system. It pursues a very long course and has an extensive distribution. In conjunction with the glossopharyngeal nerve, it is distributed to the muscles of the pharynx, larynx, and esophagus; it is also distributed to the heart, lungs, stomach, etc. In addition to its sensory and motor functions, it has vasomotor and secretory fibers.

Etiology.—This nerve may be affected by trauma at any part of its course, inflammatory conditions in the neighborhood of the nerve. It is also affected following infectious conditions, especially diphtheria, and in chronic intoxication, diseases at the base of the brain in the posterior cranial fossa, such as meningitis, tumors, hemorrhages, and vascular diseases. Diseases in the mediastinum, such as aneurysms and inflammatory conditions; operations on the neck, chest, or posterior cranial fossa; diseases of the medulla, such as bulbar paralysis; syphilis; drugs, such as atropin, aconite, etc.

Symptoms.—These depend upon the extent and the site of the lesion. If the lesion is limited to one side, then unilateral paralysis of the palate, larynx, and pharynx is the prominent

symptom. Sensory disturbances are rarely found. If the nerve is affected outside of the cranial cavity, unless the lesion occurs very high in the neck, there is, as a rule, little or no disturbance of deglutition. The symptoms arising from the heart are variable—sometimes there is a slowing and sometimes there is an acceleration of the heart action.

There are usually no respiratory disturbances, except in so far as the laryngeal involvement may interfere with respiration.

The most important symptom of unilateral involvement of the tenth nerve is laryngeal paralysis. This is characterized by paralysis of a vocal cord, which remains immovable during phonation and respiration. The voice is usually somewhat hoarse, and deep inspriation is, as a rule, accompanied by stridor. In bilateral paralysis the symptoms are very much more severe. This is generally the result of nuclear involvement. The symptoms in this condition are marked respiratory and cardiac disturbances, paralysis of the muscles of the pharynx, soft palate, and larynx, giving rise to disturbances of swallowing, marked interference with phonation, hoarse, stridulous breathing, shortness of breath, inability to cough, etc.

Among the more important branches of the tenth nerve are those that go to the muscles of the larynx; these are the recurrent laryngeals. When either or both are involved, marked symptoms occur. The recurrent laryngeal may be involved in diseases of or in operations on the thyroid gland; in diseases in the mediastinum, such as tumor or aneurysms, etc.; in intoxications and in infectious diseases, etc. The symptoms of involvement of these nerves are paralysis or weakness of the muscles of the larynx, resulting in disturbances of phonation and respiration.

Diagnosis.—The diagnosis of unilateral disease of the pneumogastric nerve is very difficult unless involvement of its recurrent laryngeal branch has taken place. The diagnosis of bilateral involvement of the nerve is made from disturbances in swallowing, in respiration, and in phonation. The diagnosis of the causative condition is made as the result of a careful consideration of the history, the course of the disease, the symptoms, and the presence of symptoms pointing to involvement of other structures.

The **prognosis** of the condition is very grave, but depends entirely upon the cause.

Treatment.—This consists in treating or removing the cause. Careful nursing, the prevention of pulmonary complications, and stimulation if necessary.

DISEASE OF THE ELEVENTH OR SPINAL ACCESSORY NERVE

This nerve innervates the sternomastoid and part of the trapezius muscle.

Etiology.—This nerve may be involved in diseases of the upper cervical segments of the spinal cord; diseases of the upper cervical vertebræ; syphilis, tuberculosis, meningitis, tumors, etc., in the posterior cranial fossa; injuries, inflammatory conditions, and tumors in the upper part of the neck; exposure to cold, and rarely toxic conditions.

Symptoms.—Paralysis or weakness of the sternomastoid and trapezius; this paralysis is flaccid in type, and there is complete or partial reaction of degeneration. The paralysis of these muscles gives rise to interference in the turning of the head, the touching of the chin to the shoulder, and the elevation of the arm above the horizontal line.

The *course* of the condition depends entirely upon the cause, and the **prognosis** is dependent upon the possibility of removal of the cause.

Treatment.—Remove the cause if possible; use massage and electricity; prevent deformity by applying appropriate apparatus. Surgical treatment is indicated in those cases in which it offers a chance for improvement.

DISEASE OF THE TWELFTH OR HYPOGLOSSAL NERVE

This nerve is distributed to the muscles of the tongue.

Etiology.—This nerve is much more frequently affected by disease within the cranial cavity than in its course outside of the skull. Diseases of the medulla, such as hemorrhage; bulbar paralysis; vascular disease; diseases in the posterior fossa, such as tumors, hemorrhages, aneurysms, fractures, necrosis of the

bone, syphilis, diseases in the upper part of the neck, such as injury, tumors, etc., are factors in the etiology. The nerve may also rarely be involved in such diseases as locomotor ataxia, multiple sclerosis, etc.; it is very rarely involved in chronic intoxications and infectious processes.

Symptoms.—If only one nerve is affected, that side of the tongue will present a flaccid paralysis, and there will be atrophy of half the tongue. If an effort is made to protrude the tongue, it will deviate toward the paralyzed side. Electric examination shows partial or complete reaction of degeneration. There is little or no disturbance of mastication and deglutition, but some disturbance of articulation. In bilateral involvement of the twelfth nerve there is inability to protrude the tongue,—it remains motionless in the mouth,—and atrophy and marked interference with mastication, deglutition, and articulation occur. In nuclear involvement there is usually also paralysis or weakness of the lips, with atrophy of the orbicularis muscle.

The **prognosis** and **course** depend upon the cause.

Treatment.—Remove or treat the cause.

DISEASES OF THE CRURA, PONS, AND MEDULLA

In the portion of the nervous system occupied by the crura, pons, and medulla there are grouped, in a very small area, many important nervous structures. Passing down through the anterior portion of these structures are the pathways for the conduction of stimuli that result in voluntary movements; in the posterior and lateral portion are the pathways that transmit upward the stimuli that are perceived as the various forms of sensation, and those stimuli that result in the maintenance of equilibrium. Situated at various levels are the cranial nerves—their superficial origin and their nuclei. On the anterior aspect of these structures are the vertebral, basilar, posterior communicating, carotids, anterior cerebral arteries and their branches.

Etiology.—Disease of this portion of the nervous system may be hereditary in character. It may also be caused by diseases of the vascular system, hemorrhage, more frequently softening

the result of thrombosis, or rarely embolism; aneurysm of the vertebral, basilar, or other vessels may compress these structures. Tumors in the posterior or middle fossa of the skull; an acute or subacute inflammatory condition that may be limited to the gray matter or affect the meninges, etc.; syphilis, leukemia; trauma; such diseases as amyotrophic lateral sclerosis, multiple sclerosis, locomotor ataxia, etc., are also among the causes of the condition.

Symptoms.—The symptoms of involvement of the medulla, pons, or crura depend upon the location, the extent, and the character of the lesion. A thorough knowledge of the anatomy and physiology of these parts is absolutely essential. (See Fig. 32.)

In those cases in which the condition is due to tumors the onset is usually more or less gradual, and is accompanied by symptoms of an intracranial tumor, *e. g.*, headache, vomiting, etc. If the disease is due to a vascular lesion, the onset may be acute, subacute, or gradual. In these cases prodromal symptoms generally occur that are caused directly by the vascular disease. Thus an attack is frequently preceded by a feeling of pressure in the head, vertigo, insomnia, tinnitus aurium, etc. The paralysis then comes on suddenly, and there may or may not be complete loss of consciousness, or very severe vertigo or convulsive seizures may occur. The distribution of the paralysis, the ataxia, and the sensory disturbance, etc., depend upon the location of the lesion. Those cases due to an inflammatory condition, or in which the bulbar symptoms are only a part of the general disease, are apt to have a slower onset, depending upon the location of the lesion.

There is generally weakness or paralysis of one side of the body, with involvement of the cranial nerves upon the opposite side, giving rise to crossed paralysis. If the lesion is in the crura, the ocular muscles of one side are affected, and the face, arm, and leg upon the opposite side (Weber's syndrome) are also involved. If the attack is accompanied by tremor of the paralyzed limbs, it is called Benedikt's syndrome. If the affection is in the pons, the functions of the fifth nerve upon one side of the body and the arm and leg upon the opposite side are af-

fected. If the lesion is in the medulla, the functions of the sixth, seventh, and eighth nerves upon one side, and the arm and leg upon the opposite side, of the body are affected—Millard-Gubler syndrome. If the lesion is more centrally situated, there may be a bilateral involvement of the structures, with disturbance of the functions of both sides of the body and of the cranial nerves corresponding to the level of the lesion. In the majority of cases, in addition to the bulbar paralysis, there is also a weakness or paralysis of the arm and leg on the opposite side to that on which the paralysis of the bulbar nerves is more marked, or, in the more severe cases, a paralysis or weakness of both sides of the body. The paralysis of the extremities is spastic in nature, with an exaggeration of the tendon-reflexes. Respiratory disturbances, increase in pulse-rate, and rise in body temperature are not unusual. In fatal cases these symptoms become more marked as the end approaches. Patients suffering from disease of these structures are usually very emotional.

Sensory disturbances may occur if the lateral and posterior columns of the medulla, pons, and crura are involved.

In those cases due to occlusion or rupture of the posterior inferior cerebellar artery the affected area lies in the lateral portion of the medulla and involves the restiform body, the motor nucleus of the ninth and tenth, the descending root of the fifth, part of the reticular formation, and sometimes the olivary body and a portion of the cerebellum. The clinical symptoms consist of unilateral paralysis of the palate and larynx, difficulty in swallowing, loss of pain and temperature sense in a portion of or the entire distribution of the fifth nerve on the side of the lesion, and a corresponding loss of pain and temperature sense, from the face downward, upon the opposite half of the body. On the side of the body on which these sensory disturbances occur tactile sense, deep pressure sense, and muscular sense are generally intact. In some cases there is also an ataxia of the arm and leg of the same side. The onset of this condition is sudden, attended with giddiness or vertigo, a tendency to fall to one side, and frequently nystagmus. Consciousness is usually not disturbed. There may be retrac-

tion of the eyeball, drooping of the eyelid, and contraction of the pupil.

Diagnosis.—Diseases involving the brain-stem must be differentiated from diseases of the hemispheres and from more diffuse conditions in which the involvement of the brain-stem is but a part of the disease. The character and the location of the lesion must also be determined. Diseases that are confined to the brain-stem are differentiated from diseases involving the hemispheres by the fact that if conduction pathways are affected in brain-stem conditions, a crossed paralysis occurs; that is, the cranial nerves are affected on one side of the body, and the arm and leg upon the opposite side of the body. Also in brain-stem involvement the cranial nerve palsy is flaccid, whereas that due to involvement of the hemisphere is of a spastic nature.

In *lesions of the hemispheres* causing paralysis the eyes are turned toward the non-paralyzed side; in brain-stem lesions the eyes are turned toward the paralyzed side. In irritative lesions of the hemispheres the eyes are turned toward the affected side; in brain-stem conditions the eyes are turned toward the unaffected side.

The Character of the Lesion.—Tumors usually cause symptoms that come on gradually and give rise to both general and focal symptoms. The general symptoms are those of cerebral tumors, that is, headache, vomiting, vertigo, optic neuritis, etc.; the focal symptoms are those caused by pressure upon the cranial nerves and the conduction pathways, causing a crossed paralysis. It is also important to determine whether the tumor is situated within the substance of the brain-stem or whether it is situated externally. Tumors within the substance of the brain-stem generally occasion bilateral symptoms of a motor and sensory nature, with bilateral involvement of the cranial nerves which originate in the neighborhood of the tumor. There may be a predominance of symptoms on the side of the tumor, with more marked evidence of motor and sensory symptoms upon the opposite side.

In tumors situated external to the brain-stem the course is slowly progressive, beginning with symptoms that point to a

very limited involvement of the brain-stem, and that gradually progress and give rise to the characteristic crossed paralysis (Fig. 116). If the tumor is situated in the middle line, it is practically impossible to distinguish it from a tumor situated within the substance of the brain-stem.

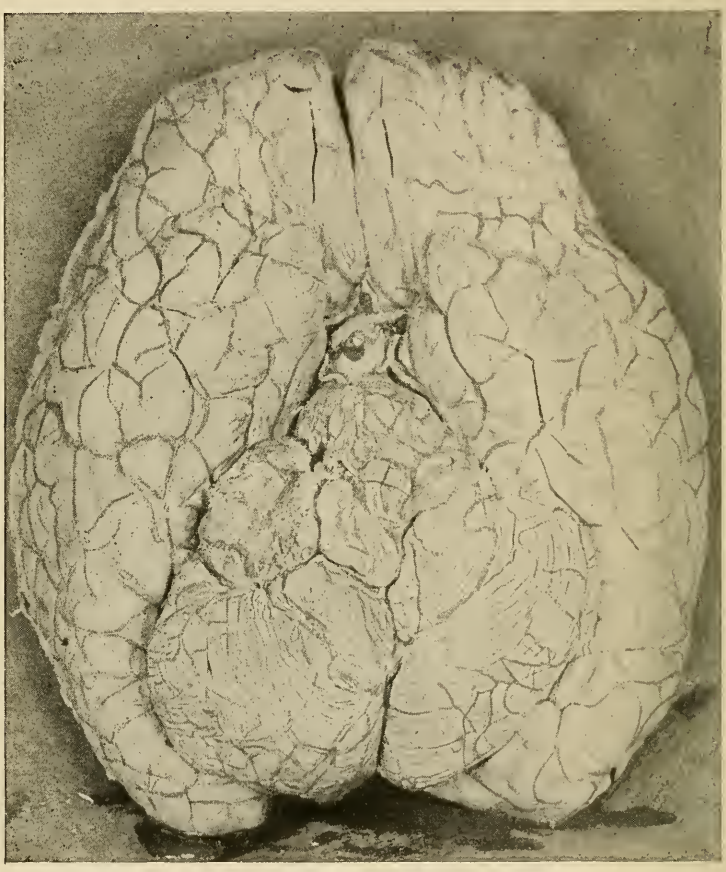


Fig. 116.—Tumor of the cerebellopontile angle.

Vascular diseases are more or less sudden in their onset; thus hemorrhage and emboli appear suddenly, whereas thrombosis occurs more gradually. When the condition is due to a thrombosis, the prodromal symptoms generally occur that are indicative of disease of the cerebral vessels. The attack itself

is usually preceded by paresthesia in the limbs about to be affected, the paralytic symptoms then appearing gradually.

If the condition is due to an *aneurysm*, the symptoms of vascular disease occur along with symptoms that are similar to those occasioned by a tumor.

If the symptoms pointing to brain-stem involvement are but a part of the manifestation of a more diffuse disease, there usually occur other symptoms that render the recognition of the underlying disease possible. In all cases the Wassermann test of the blood and spinal fluid and a careful blood and urine examination should be made.

The location of the lesion is determined by the symptoms. Lesions in the crura and upper pons generally involve the third and fourth nerves; lesions in the pons and upper medulla involve the sixth and seventh nerves; those in the lower medulla, the ninth, tenth, eleventh, or twelfth nerves. In addition to the cranial nerve involvement, depending upon the extent of the lesion there will also be involvement of the conducting pathways: if the lesion is situated anteriorly, the motor pathway is involved; if the lesion is situated somewhat more posteriorly, sensory pathways will be involved; and if the lesion is situated still more posteriorly, the pathways of coördination and equilibrium are affected. If the lesion is limited to one side of the brain-stem, the cranial nerves on the same side and the extremities upon the opposite side are affected. If the lesion is centrally placed and is large enough, the cranial nerves on both sides, originating in the location of the lesion, are affected, and according to the size of the lesion there will occur disturbance of motion, sensation, etc., in parts of the body below the lesion.

The **course** and **prognosis** of these affections depend upon the cause: in those cases due to compression by tumors, aneurysm, etc., the course is generally progressive, and unless the condition can be relieved by surgical or medical treatment, the prognosis is unfavorable. In those cases due to rupture or occlusion of the vessels the course and prognosis depend upon the severity of the symptoms. With the exception of those cases in which death occurs within a few days from bronchopneumonia and paralysis of respiration and of the heart, the course is usually

regressive—the symptoms having reached their greatest intensity at the onset of the disease, gradual improvement takes place that may end in recovery. In nearly all cases there are residual symptoms. The prognosis of diseases of the medulla is, as a rule, very serious, for, in addition to other dangers, there seems to be an unusual susceptibility to pneumonia.

Treatment.—The cause should be sought and treated. If there is the slightest suspicion that syphilis is at the base of the condition, antiluetic measures should be instituted. In those cases that are due to vascular disturbances the patient is to be placed at absolute rest; if the blood-pressure is low, it is advisable to administer strychnin; if the blood-pressure is high, the patient should be freely purged and ice-bags applied to the neck in the suboccipital region. In those cases due to inflammatory conditions counterirritation and cold applications to the suboccipital region, free purgation, and free elimination by the skin and kidneys are to be encouraged, and the patient is to be kept absolutely quiet. In all these cases careful attention must be given to the diet, for, owing to the difficulty in swallowing and the loss of laryngeal and pharyngeal reflexes, there is great danger of food passing into the bronchi. In some cases it is not only advisable, but absolutely necessary, to use a feeding-tube.

PROGRESSIVE BULBAR PARALYSIS

This condition, which is also known as *glossolabiopharyngeal paralysis*, is a chronic progressive disease involving the nuclei of the motor cranial nerves, usually beginning with the twelfth, and slowly progressing upward and involving the nuclei on both sides of the medulla. In very rare cases it begins high up in the pons or medulla, and proceeds downward. In some cases the bulbar symptoms are a part of a more wide-spread affection, such as amyotrophic lateral sclerosis, multiple sclerosis, etc.

Etiology.—The cause of this disease is unknown. In some cases it occurs in early life, when it is usually a congenital condition, affecting several members of a family. It generally appears between the thirtieth and the sixtieth year, although no age is exempt. Injury, emotional disturbances, overwork of the muscles of the tongue, lips, and palate, such as occurs in

individuals who play on wind-instruments, glass-blowers, etc.; exposure to cold; chronic lead-poisoning, etc., have all been held responsible for this disease.

Pathology.—There is a progressive atrophy of the motor neurons, involving the nuclei of the twelfth and the eleventh, tenth, ninth, seventh, fifth, and sometimes of the sixth and third nerves. In addition to degeneration of the motor cells and their processes, there also occur atrophy and degeneration of the muscles with which these cells are connected.

Symptoms.—The disease usually first manifests itself by difficulty in speech, the articulation of words containing the linguals being interfered with. As the disease progresses this difficulty in speech increases, until finally it is almost impossible to understand these patients. With the progress of the speech disturbances there may be noticed fibillary tremors in the tongue, lips, pharynx, etc.; these are later followed by atrophy of the affected structures. At first the patient's speech difficulty manifests itself only after prolonged conversation, but later it is constantly present. Disturbances of the acts of chewing and swallowing are also observed. The patient finds it difficult to pass the food from the mouth into the pharynx with his tongue; it is also noticed that fluids regurgitate into the nose or pass into the larynx, bringing on fits of coughing. Finally, there is complete inability to swallow solid or liquid foods. Later on the muscles of mastication become affected, and the lower jaw is not held in apposition with the upper, and the mouth remains open. Eventually the mastication of food becomes impossible.

When the facial muscles are involved, there is an absence of facial expression and the facial folds become obliterated; the lower lip hangs down, and there is more or less constant dribbling of saliva. Phonation and respiration finally become involved, and the voice is weak and monotonous and without modulation. Toward the end of life respiration becomes difficult and irregular, and attacks of suffocation occur. Physical examination discloses weakness or paralysis of the tongue, facial muscles, etc., which is followed by fibrillary tremors and atrophy. The pharyngeal and laryngeal reflexes are usually absent. Laryngoscopic examination shows at first weakness of the adductors,

which later become paralyzed. In this disease the weakness of the muscles always precedes the appearance of the atrophy. Electric examination reveals a partial reaction of degeneration, which usually becomes complete only in the later stages of the disease. It is only in very exceptional cases that the paralysis advances to the ocular muscles. These patients are, as a rule, very emotional; they emaciate rapidly, this probably being due to the interference with nutrition.

Course.—The disease usually advances slowly over a period lasting from one to three or four years. During this time remissions may ocur, but these are generally of short duration.

The **prognosis** is extremely grave, the patient succumbing to inanition, bronchopneumonia, asphyxia, or some intercurrent disease.

The **diagnosis** usually presents no difficulty. The disease must be differentiated from *vascular diseases* of the cerebral axis: in this last condition the onset is generally much more sudden, and there are present symptoms of vascular disease, evidence that the lesion is not limited to the nuclei of the motor cranial nerves, and the condition is usually not bilateral without the existence of symptoms pointing to extensive involvement of the cerebral axis. It is differentiated from *tumors* situated in the neighborhood by the fact that in the latter there are usually some of the general symptoms of brain tumor; the development of symptoms is, as a rule, much slower, and there is commonly evidence of extensive involvement of the cerebral axis, which manifests itself as a spastic condition of the extremities; and the symptoms are not symmetrically bilateral. From *multiple sclerosis* the disease is distinguished by the spinal and cerebral symptoms. From *amyotrophic lateral sclerosis* it is differentiated by the presence of spinal symptoms. From *multiple neuritis* in which the cranial nerves are involved, especially in postdiphtheric paralysis, it is distinguished by the history, by the comparatively rapid onset and rapid progress of the symptoms, by the distribution of the paralysis, and by the occurrence of other symptoms, such as sensory disturbances and involvement of the extremities. It is to be differentiated from *pseudobulbar paralysis*, in which condition bilateral lesions in

the lower part of the precentral convolutions, or in the motor pathways from these centers to the cerebral axis, bring about bilateral paralysis of the tongue and face. This may be due to a congenital defect, multiple sclerosis, or several apoplectic attacks. In this condition a differential diagnosis is made with comparative ease by the absence of atrophy and of fibrillary tremors; the history of the onset; the presence of symptoms pointing to some vascular or other disease, and the usual presence of aphasia. From *myasthenia gravis* it is differentiated by the fact that in the latter condition there is more or less general muscular fatigue, which appears rapidly upon exertion; the frequency with which the ocular muscles are affected; the absence of fibrillary tremors; the presence of the myasthenic reaction, and the frequency of intermissions when the patient seems to be almost entirely well.

Treatment.—The most important factor in the treatment of this conditon is the maintenance of nutrition of the patient. This is best accomplished by giving nutritious food in as small a bulk as possible, to be followed later by semisolid food. If necessary, a feeding-tube should be used. The patient's general health is to be maintained by an abundance of fresh air, general massage, hot and cold sponge-baths, etc. Strychnin in proper doses, in conjunction or alternating with tonics, the combination of iron, quinin, and strychnin, etc., are indicated. Water should be given freely. Great care should be exercised in feeding the patient so as to prevent food or liquids entering the respiratory passages.

DISEASES OF THE SPINAL CORD

Owing to the anatomic construction and physiologic functions of the various portions of the spinal cord, this structure may be affected pathologically in several ways. Disease processes may be limited more or less exclusively to the gray matter; they may be confined to the tracts, or they may be more or less diffuse, involving both the gray and the white matter.

ACUTE ANTERIOR POLIOMYELITIS

This condition, also known as infantile spinal paralysis or atrophic spinal paralysis, is an acute infectious disease seen chiefly among children, but also affecting adults. It occurs in epidemics or sporadically.

Etiology.—This disease is seen most frequently in the first five years of life. It is more common during the later summer months than at any other period of the year. Exposure, overexertion, and trauma may predispose to the condition. It may also occur in connection with various infectious diseases, such as measles, scarlet fever, pneumonia, etc. The specific cause of the disease is a microörganism that probably enters the system through the nasal mucous membrane.

Flexner has shown that the secretion from the mucous membrane of the nose and mouth contains the infective organism. The period of incubation is from a few days to two weeks. The disease may be carried by persons not affected. Epidemics of the disease have frequently occurred, and it has usually been possible to trace the transmission of the affection from person to person or from place to place.

Pathology.—Examination of the spinal cord of a person dying during the acute stage of the attack shows an active congestion and inflammation of the meninges and of the white and gray

matter of the spinal cord, and sometimes of the brain. The congestion is more marked in the anterior part of the gray matter, on account of its greater blood supply. In some cases the pons, medulla, and crura cerebri are also involved along with the spinal cord. The degree of inflammation in the meninges varies with the severity of the disease. It is probable that in cases of the abortive type there is merely a congestion in the distribution of the anterior spinal artery, which rapidly disappears and leaves no permanent symptoms. As the result of congestion and inflammation of the spinal cord and meninges these structures are the seat of numerous small hemorrhages, infiltration, and emigration of leukocytes. The leukocytes and many small round cells infiltrate the tissues. In the more severe cases the nerve-cells undergo degeneration and are absorbed. In some cases degeneration does not go on to complete destruction and recovery is possible. The neurogliar elements also undergo destructive changes varying in extent with the severity of the lesion. As the result of atrophy and sclerosis there is a decrease in the size of the anterior horn. When the gray matter which lies close to the central canal in involved, there is, in addition to the paralysis, marked disturbance in the growth of the limbs, this portion of the gray matter apparently bearing a close relation to the growth of the tissue. Early in the disease, before the development of the paralysis, the spinal fluid contains lymphocytes, both large and small, and polynuclear leukocytes, and it is more or less cloudy in appearance. As the paralysis develops this condition disappears.

Symptoms.—The disease usually begins acutely, the temperature ranging from 99° to 104° F. Along with the fever there are general constitutional disturbances, such as loss of appetite, vomiting, intestinal disorders, stupor, delirium, and sometimes convulsions. In some cases these symptoms are accompanied by stiffness and pain in the back, headache, etc., closely resembling meningitis. During this stage, which may last from a few hours to several days, there is considerable prostration and often more or less pain and tenderness of the extremities. At this time it is usually impossible to determine the nature of the illness. Toward the close of this stage the

paralysis or weakness, which is wide-spread, is first noticed. If the fever is mild, it may be overlooked, the paralysis being the first symptom noticed. The paralysis may at first involve all four extremities, but more commonly it affects one extremity, either an arm or a leg, or both arms, or both legs and one arm, etc. In some cases the face or ocular muscles may be involved, but this is rare. Not infrequently, in addition to the involvement of the extremities, the trunk muscles may be affected (Figs. 117, 118, 119).

At the onset the paralysis is usually more extensive in distribution than it is destined permanently to be. It is always flaccid in type, and as time passes marked atrophy of the affected muscles takes place. The electric reaction depends upon the extent and the severity of the involvement. Within a short time after the attack has passed off there is a return of power in the muscles that were least affected, and they contract to voluntary impulses, and although the paralysis may have been very extensive at the onset, it may finally be limited to a very few muscles. Frequently the paralysis may involve only parts of several muscles.

The tendon-reflexes associated with the affected muscles are either lost or markedly decreased. Pain is not a permanent symptom, but in a few cases it is so severe that the condition may be mistaken for rheumatism. Sensibility is not diminished; skin reflexes are not disturbed, except in so far as they are interfered with by the presence of the paralysis.

The Electric Reaction.—In those muscles that are least affected there is usually a response to both faradic and galvanic current. In those muscles that are more severly affected, there is complete reaction of degeneration, and if the muscle continues to atrophy and the muscle tissue to disappear, there finally occurs complete loss of all electric contractility.

Vasomotor Trophic Symptoms.—In many cases the circulation in the affected limb is impaired and the limb feels cold and presents a bluish appearance. When the affection has been at all severe, and in fact in practically all cases, there is some arrest of growth as well as deformity. The affected limb is tardy in development, and this defect involves both the soft and the bony

structures of the limb. Bowel and bladder control is not affected. The deformity arises in consequence of the con-

Fig. 117.—Anterior poliomyelitis. In the acute stage in this case there was complete paralysis of the muscles of the trunk, neck, upper and lower extremities. At the present time there remains weakness of the neck muscles and of the muscles which fix the scapulæ to the chest walls.

Fig. 118.—Anterior poliomyelitis, shoulder type right side, leg type left side.

tracture of the antagonists to the paralyzed muscles, the contraction gradually resulting in shortening. Flail-joints result when the muscles that support the structures at a joint are

the seat of paralysis, and occur more often at the hip and shoulder. The distribution of the paralysis varies greatly in different cases. When the paralysis affects the lower extremity, two

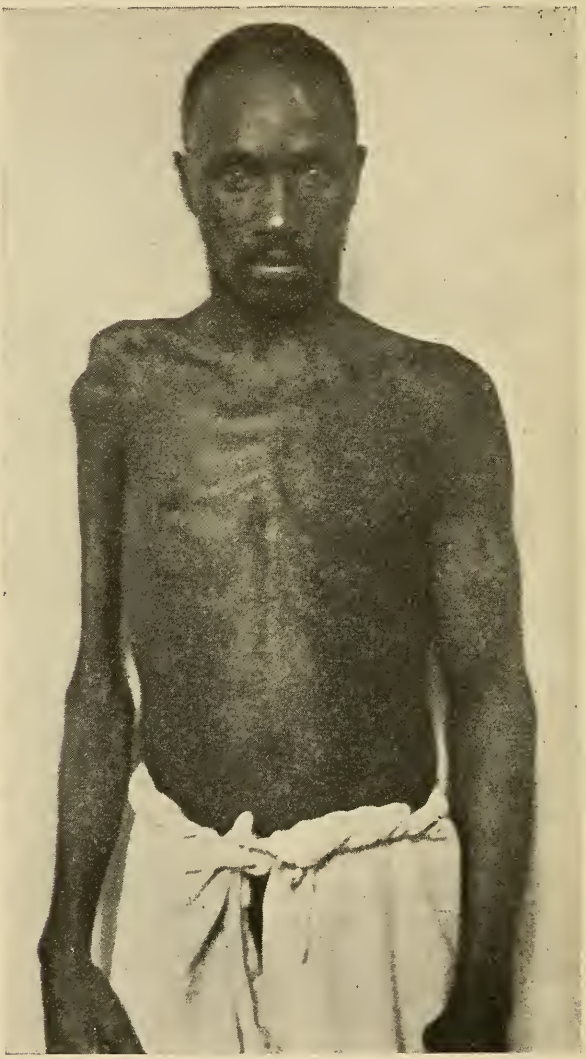


Fig. 119.—Anterior poliomyelitis.

types of disease may be recognized: the *leg type*—the paralysis involving some or all of the muscles on the anterior or anterior and posterior aspect of the leg, bringing about interference with

the movements of the foot. In some cases there is foot-drop, with ability to move the toes. If the paralysis is persistent, deformity eventually occurs. In the *thigh type* the muscles about the hip-joint are chiefly affected, those on the inner part of the thigh and below the knee often escaping. It is in these cases that we have the flail-joint at the hip. In some cases all the muscles of the lower extremities may be involved.

Two types of affection are also observed when the arm is involved: in the *lower arm type* the muscles below the elbow are involved, whereas in other cases the muscles of the hand are affected; in the *upper arm type* the muscles about the shoulder and arm are involved. In many cases along with the involvement of the extremities there is also affection of the muscles of the trunk—rarely, of the muscles of the trunk alone.

The paralysis is rarely symmetric (Fig. 118). The chief characteristics of this form of paralysis is that it is atrophic; that distribution of the paralysis bears no relation to the arrangement of the muscles in the limb, to the action of the muscles in producing movement, or to the nerve-supply, but is wholly dependent upon the arrangement of groups of cells in the anterior horns of the spinal cord which control muscles or parts of muscles.

Diagnosis.—This condition must be differentiated from *febrile states*, accompanied by immobility of one or more of the extremities, from rheumatism, rickets, osteomyelitis, fractures, etc. In these affections a proper examination will always disclose the fact that pain is the cause of the immobility of the limb, and that when an effort is made to move the part, muscular contraction can be observed. In addition to this the electric reaction and the presence of the deep reflexes will make the diagnosis clear.

From *multiple neuritis* the condition is differentiated by the facts that multiple neuritis occurs very rarely in childhood; does not attain full development so quickly; the feverish stage is apt to last longer; pain is more marked, the nerves and muscles being tender to pressure; disturbance of sensibility is usually present; the distribution of the paralysis is confined to muscles supplied by the peripheral nerves.

From *infantile cerebral paralysis* this condition is differentiated

by the distribution of the paralysis, which is more apt to be hemiplegic or diplegic in type; the paralysis is spastic, and the reflexes are increased. From *birth palsy* or a *localized injury* of the brachial plexus the disease is differentiated by the history of the case. *Hemorrhage into the spinal cord in children* is extremely rare; if the hemorrhage is limited to the gray matter, the symptoms will resemble those of the anterior poliomyelitis, except for the very acute onset and the absence of fever. In *myelitis* the involvement of the spinal cord is usually diffuse, and in addition to the symptoms of involvement of the gray matter there is disturbance of sensibility, of motion, and of control of the bowels and bladder, due to interference with the transmission of the impulses up and down the cord. If we have reason to suspect the existence of poliomyelitis, examination of the spinal fluid, made on the second or third day of the febrile condition, before the paralysis has appeared, makes a diagnosis possible. Cerebrospinal fluid is increased in amount, is opalescent, and contains a large number of small and large lymphocytes and a few polymorphonuclear leukocytes, and the fluid gives a protein reaction to the butyric-acid test.

Prognosis.—The prognosis is favorable as regards life. In those cases that point toward involvement of the upper cervical region or brain-stem, however, the prognosis as to life is very grave. The prognosis as to complete recovery is most unfavorable. In some epidemics many cases occur in which recovery is complete. In the majority of cases marked improvement may be looked for. The more circumscribed the paralysis at the onset of the attack, the less permanent will the disturbance of function be. As a rule, a limb that is affected never entirely regains its power, and usually shows atrophy and shortening. The electric examination is of assistance in making a prognosis: muscles that respond to the faradic current after three weeks usually recover completely; those that fail to react to this current may improve, but will not recover completely. Improvement extends over a period of from six months to two years. The prognosis as to recovery of function is somewhat affected by secondary changes, such as contractures and interference with growth.

Treatment.—During the initial or febrile stage the patient

should be kept absolutely quiet in bed, and active movements prevented as far as possible. Mild counterirritation may be made along the spine, and the application of dry cups or leeches to the back. The gastro-intestinal tract should be cleared with a mild purgative, and diaphoresis induced by warm drinks and hot applications. If the temperature is high, sponging with alcohol is advisable. If the child is in great pain or there is marked restlessness, the bromids, with or without codein, may be given. The diet should be light, and the patient should be persuaded to drink as much water as possible. Owing to the fact that this is a contagious disease, the child should be completely isolated, and the nose and throat sprayed with a mild antiseptic solution every few hours; this should be continued for several weeks after the fever has subsided.

During the acute stage sodium salicylate in appropriate dose may be given. Usually the constitutional symptoms disappear at the end of a week or ten days, and the paralysis, which was very extensive at first, shows evidence of improvement and becomes limited to a smaller area. At this stage it is advisable to give strychnin in proper doses, and to encourage the child to use those portions of the body that can be moved; this last can sometimes be accomplished by placing the child in a tub of warm water and encouraging it to play. These warm baths must, of course, be given under proper supervision, and the patient must not be permitted to remain in the water longer than fifteen minutes. They may be given daily.

Deformities are to be prevented as far as possible by supporting the limbs and dependent portions of the body, and by massage and passive exercise. Small doses of potassium iodid or syrup of iodid of iron seem to be of benefit. Skilful massage, gymnastic exercise under proper supervision, and the use of electricity will frequently assist in the recovery of muscles that would otherwise not regain their functions. This treatment should be persisted in for from one to two years. The general health and physical condition of the patient should be carefully looked after. Surgical measures and the various forms of orthopedic apparatus may restore, at least partially, the functions of parts of the body that do not respond to medical treatment.

SUBACUTE AND CHRONIC ANTERIOR POLIOMYELITIS (PROGRESSIVE SPINAL MUSCULAR ATROPHY)

This is a rare form of spinal cord disease in which there is a progressive development of paralysis due to a slowly advancing atrophy of the primary motor neurons of the cord.

Etiology.—The disease generally affects adults. Trauma, toxic substances, and disturbance of the lymphatic circulation are believed to be among the causative agents.

Pathology.—Examination of the spinal cord of an individual who has suffered from this disease shows an atrophy of the anterior roots and a shrinkage of the anterior horn cells in different regions of the cord. The changes in the cells vary from a simple decrease in size of the cell and its nucleus to an almost complete disappearance. In addition to the atrophy of the cells, there are also atrophy and finally disappearance of the dendrites and axons. As a consequence of this degeneration there is atrophy of the anterior root-fibers and of the nerves to their termination in the muscles. The affected muscles also undergo changes in which there is a simple atrophy of the muscle-fibers.

Owing to the fact that the lesion may begin in different parts of the spinal cord, three clinical types of this disease are encountered. In the first type, atrophy begins in the cells in the lower portions of the spinal cord, gradually extending upward, but not involving the arms. In the second type, known as Duchenne's subacute ascending paralysis, the atrophy begins in the cells in the lower part of the cord, and gradually advances upward until all the motor cells are involved. In the third type, the Aran-Duchenne type of progressive atrophy, the atrophic process begins in groups of cells in the lower part of the cervical or the upper dorsal region of the cord, and as the disease advances the upper cervical segments become affected, and later the lumbar region is involved.

Symptoms.—The symptoms of these three types of this disease vary only in that different regions of the cord are primarily affected. In chronic ascending paralysis there is a slowly advancing flaccid paralysis, beginning in the muscles that flex

the foot and extend the toes, first one leg, and then, within a few months the other leg, being affected. The paralysis and atrophy gradually become more evident until finally a fully developed foot-drop appears. Along with the paralysis and atrophy there is usually a fine fibrillary tremor in the affected muscles. As the disease progresses the adductors of the thigh and the glutei muscles become affected, and difficulty in going up steps and in rising to the erect position after sitting ensues. At about the same time that these muscles are involved the muscles of the posterior aspect of the leg and thigh also become affected. The extent of paralysis corresponds to the degree of atrophy present. There are no sensory disturbances, and no interference with bowel and bladder functions. The reflexes and the electric reactions in the affected muscles depend upon the amount of muscle tissue that is unaffected. In the first type of cases the disease may not advance beyond the involvement of the legs and thighs, the patient living for years in a state of paraplegia.

In the second type of cases the disease begins in the same way as in the first type, progresses upward, the muscles of the back, trunk, and finally of the shoulder and hands becoming involved. If the patient does not die of some intercurrent condition, the motor cranial nuclei become involved.

In the Aran-Duchenne type the muscles that move the thumb are the first to become affected. As the condition advances the small muscles that move the fingers become involved, making the finer movements of the fingers clumsy or impossible. As the atrophy progresses there is a flattening of the thenar and hypothenar eminences, and hollows are produced between the metacarpal bones. Owing to paralysis of the interossei the first phalanges of the fingers cannot be flexed, and as there is no opposition to the long flexors and extensors of the fingers, the hand assumes a characteristic position in which it resembles a a claw. As a rule, one hand is involved before the other. There is a tendency to gradual extension of the paralysis and atrophy to the shoulder and then to the muscles of the arm, until the entire arm and forearm are completely paralyzed. The disease may then involve the muscles of the neck and of the trunk, and

if the patient does not die from some intercurrent affection the legs finally become involved.

The characteristic symptoms of this disease are paralysis and atrophy, the extent of the paralysis depending upon the amount of atrophy present; fibrillary tremors occurring in the paralyzed muscles; disturbance of the electric reaction, depending upon the amount of muscle tissue that remains intact: at first there is partial reaction of degeneration, later complete reaction of degeneration, and finally loss of electric response occurs. The deep reflex reaction also depends upon the amount of muscle tissue remaining intact, there being a gradual diminution of the reflex response as the muscle atrophies.

Course and Prognosis.—The duration of the disease varies from two to many years. Spontaneous arrest of atrophy may occur at any time during the course of the affection, but the prognosis is serious, as there is a tendency for the disease to go on to extreme atrophy, death ensuing as the result of respiratory involvement or bulbar paralysis. The three types described show the usual form of progress of the disease, but cases occur that do not conform to these types in this respect.

Diagnosis.—This disease must be differentiated from amyotrophic lateral sclerosis, muscular dystrophy, syringomyelia, and multiple neuritis.

In *amyotrophic lateral sclerosis* the increase of the knee reflexes appears early, with the development of the Babinski reflex; the paralysis is of the spastic type; the progress of the case is usually more rapid, and the bulbar symptoms either precede the affection of the limb or occur early in the disease. In this affection there is a combination of the symptoms of a progressive muscular atrophy and of lateral sclerosis.

In *muscular dystrophy* there is an absence of fibrillary tremors, and the appearance, frequently, of a hypertrophy or pseudohypertrophy. The disease also develops usually in childhood.

In *syringomyelia* there are early appearance of sensory disturbances of a characteristic nature,—a loss of sensibility of pain, temperature, with retention of tactile sensibility; the onset of trophic symptoms, and, usually, increase of the knee reflexes.

In *multiple neuritis* the presence of sensory disturbances, the

loss of deep reflexes, and the absence of fibrillary tremors and presence of superficial vasomotor and trophic disturbances will serve to differentiate the condition.

Treatment.—The treatment of this affection resolves itself into looking after the general health of the patient. He is to be given wholesome food, to maintain the general nutrition, fresh air, and a very moderate amount of exercise. Fatigue of the affected muscles is to be absolutely avoided, and infections of all kinds guarded against. General massage and passive exercise are of value in so far as they promote the circulation, thereby increasing the nourishment of the muscles. Strychnin in proper doses is to be given three or four times a day, and arsenic and other general tonics will be found beneficial.

Deformity should be prevented, and suitable apparatus may be worn to assist the patient in his activities.

AMYOTROPHIC LATERAL SCLEROSIS (CHARCOT'S DISEASE)

This disease usually develops in adult life. Very little is known as to its cause, some assuming that there is a congenital predisposition to weakness of the motor elements of the nervous system.

Pathology.—The spinal cord shows degeneration and atrophy of the pyramidal tracts and of the anterior horns, but no involvement of the sensory pathways. In the anterior horns there is a marked degeneration of the large motor nerve cells and their processes. In most cases it is the cervical enlargement that is principally involved. In the medulla and pons there is an atrophy of the pyramidal tracts and of the motor nuclei of the twelfth, the seventh, and frequently the tenth, eleventh, and fifth nerves. In some cases the atrophy of the pyramidal tracts can be traced as high as the precentral convolution.

Symptoms.—This disease is characterized by three sets of symptoms—those of chronic anterior poliomyelitis, of lateral sclerosis, and of bulbar paralysis. The onset is usually marked by weakness and atrophy in the upper extremities, or weakness and stiffness in the lower extremities. As a rule, one arm or

one leg is more markedly affected than the other. The paralysis and atrophy gradually extend and become more severe, and at the same time muscular rigidity increases. The disease is slowly progressive, and within a year both arms and legs may be in a state of advanced paralysis. Fibrillary twitchings are usually present, and may precede the paralysis. There is a complete absence of sensory symptoms. The tendon reflexes are markedly increased, and motor weakness is generally combined with atrophy and contractures. The disease usually begins with fibrillary twitchings, which may be more or less diffuse. These may be followed either by stiffness in the legs or by weakness in the upper extremities, the weakness affecting the smaller muscles of the hand. The weakness is followed by atrophy, producing a deformity that resembles "claw hand." Contractures develop, and the arms are strongly adducted, with the forearm flexed and the hand pronated (Fig. 120).

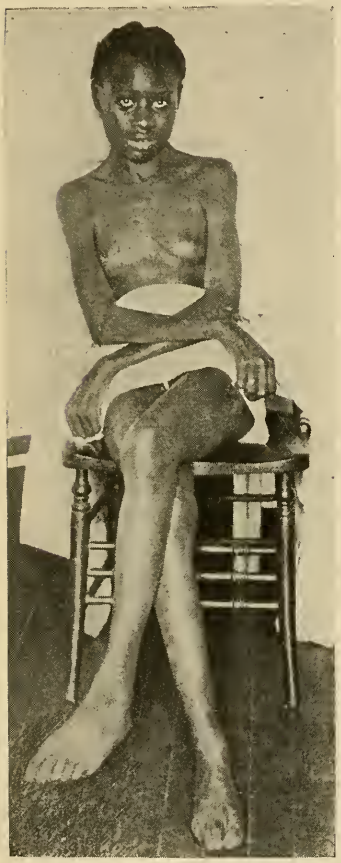


Fig. 120.—Amyotrophic lateral sclerosis, showing atrophy of the small muscles of the hand, spastic condition of the lower extremities.

In the lower extremities there is usually an absence of atrophy, but a marked rigidity appears early. The *Babinski sign* may or may not be present. The gait is slow and of the spastic type. In the later stages of the disease the atrophy also affects the legs. In some few cases the disease begins with atrophy in the lower extremities, the condition gradually extending upward. There are no bowel or bladder disturbances. Later on in the disease the symptoms of bulbar paralysis develop. In some cases bulbar paralysis is the first symptom to manifest itself,

the rigidity in the lower extremities and the involvement of the upper extremities appearing later and clearing up the diagnosis. When the bulbar paralysis occurs, it manifests itself first in disturbance of speech, the patient at first speaking indistinctly, this giving place finally to an anarthria. At about the same time, or shortly thereafter, difficulty in swallowing and in the mastication of food is noticed. This form of bulbar paralysis may be preceded by rigidity of the facial muscles and by an increase of the jaw-jerk. As the disease advances the face becomes expressionless, the lower jaw drops, and saliva trickles out between the lips. Speech now becomes unintelligible, and swallowing is no longer possible. Usually the patient becomes emotional, laughing and weeping readily.

Differential Diagnosis.—It is distinguished from *chronic anterior poliomyelitis* by the presence of spastic symptoms, along with the atrophic paralysis, and in typical cases the distribution of the symptoms. From *multiple sclerosis* it is differentiated by the absence of nystagmus, atrophy of the optic nerve, peculiar speech disturbance, intention tremors, etc. From *compression of the spinal cord in the cervical region* the differentiation is made by the absence of sensory disturbances and of bowel and bladder disorders.

Course and Prognosis.—The *course* of this disease is usually progressive, lasting from two to four years, although some cases may be of longer duration. The *prognosis* as to life is absolutely hopeless.

Treatment.—This is limited to making the patient as comfortable as possible. Where there is difficulty in swallowing, feeding by tube will be necessary in order to maintain the general nutrition. The stiffness may be relieved by massage, passive movement, and prolonged warm baths. Sedatives are indicated to relieve the muscle rigidity and frequently to quiet the patient.

SYRINGOMYELIA AND SPINAL GLIOSIS

Syringomyelia is a condition characterized by the formation of a cavity within the spinal cord; *spinal gliosis* is a condition in which a new-growth takes place in the substance of the spinal cord, without leading to a material increase in the size of this organ. Cavities of the cord are frequently produced by disintegration in this form of new-growth. For present purposes both conditions may be considered together.

Etiology.—*Cavity formation* has been ascribed to congenital dilatation of the central canal, secondary to a central myelitis, disintegration of tumors, or a gradual enlargement of a congenital cavity in the cord. *Glial tumors* have been shown to be the result of nests of glia-cells left behind in the developmental period in the neighborhood of the central canal or in the region of the posterior septum. These nests tend, either spontaneously or under the influence of trauma, to proliferate, and by their increase to form longitudinal tumors.

Pathology.—The spinal meninges are usually normal. The cord either appears unaltered or slightly larger or irregular in outline. On section of the cord a cavity of varying size is seen. In other cases section of the cord reveals the presence of a tumor of varying size and shape, occupying the center of the cord, and involving to a certain extent the gray matter. The tumor or cavity usually extends longitudinally, and varies as to the extent of its longitudinal growth and the amount of gray matter that is involved. Microscopic examination of the tumor shows that it consists of glia-cells and fibers. The cavities are usually lined by a firm, compact membrane in which a layer of endothelium can sometimes be recognized.

Symptoms.—The condition is characterized by the presence of three characteristic groups of symptoms, which are present in the majority of cases: (1) Disturbance of sensation: there is usually a loss of sensibility to pain and thermal sense, with the retention of tactile and muscle sense; this condition is known as dissociation of sensation; patients so afflicted frequently receive injuries or burns without experiencing pain, although the sense of touch is retained. The distribution of the sensory disturbance

depends upon the location of the lesion in the spinal cord and upon its extent.

(2) Trophic disturbances occurring in the skin, muscles, bones, and joints: in many cases these disturbances are probably due to trauma, which has passed unobserved because of the loss of the sense of pain, and they have, therefore, been neglected. In some cases, however, this is not the case, and the trophic disorders are due to disturbance or destruction of the trophic centers in the spinal cord. There may be interference with the perspiration, the part being either abnormally covered with sweat or abnormally dry. Superficial or indolent ulcers may form on the skin.

Morvan has described a disease in which painless felons appear upon the fingers, these being frequently associated with other trophic disturbances of the skin and nails. This disease is believed to be a form of syringomyelia.

Bone necrosis, small abscesses upon the fingers, ankylosis of the finger-joints, abnormal growth of the nails, spontaneous fractures due to trophic bone changes, affections of the joints similar to that type described by Charcot as developing in locomotor ataxia, etc., occur with comparative frequency.

(3) Progressive muscular atrophy with paralysis: this muscular atrophy usually begins in the hands, and less frequently in the muscles of the shoulder. As a rule, the small muscles of the hand are the first to become atrophied, and the claw-hand position develops.

The atrophic muscles generally show fibrillary tremors. This atrophic paralysis usually progresses very slowly, and one side of the body commonly shows greater involvement than the other. Later on the muscles of the trunk, and finally those of the lower extremities, undergo atrophy, and along with this paralytic symptoms develop. In those cases in which the lateral tracts are compressed spastic paralysis with contractures and deformities develops. Unless the lumbar region is involved, there is usually no disturbance of the sphincters. Paresthesias are generally present, and some patients complain of considerable pain. Among the unusual symptoms of this disease is the evidence of involvement of the cranial nerves, this, of course,

occurring when the brain-stem becomes involved in the pathologic process. The distribution of both the motor and the sensory symptoms corresponds to the spinal cord segments, and not to the distribution of the peripheral nerves. The lower cervical and upper dorsal regions of the cord are most frequently affected, and as these segments of the cord contain the centers of the cervical sympathetic, symptoms of paralysis of the sympathetic of one or of both sides are usually present; these consist of retraction of the eyeball, sluggish pupillary reaction with loss of the sympathetic pupil reflex, narrowing of the palpebral fissure, and defective secretion of sweat.

Diagnosis.—From *progressive muscular atrophy* of spinal origin this disease is distinguished by the presence of the characteristic sensory disturbances.

From *locomotor ataxia* it is distinguished by the absence of the characteristic pupillary and bowel and bladder disturbances, by the presence of dissociation of sensation, muscular atrophy, and paralysis.

From *caries of the cervical spine* it is differentiated by the presence of dissociation of sensation, muscular atrophy, and paralysis.

Course and Prognosis.—The disease usually manifests itself between the twenty-fifth and the forty-fifth year. Its progress is generally very slow, and it may extend over many years. Remissions occasionally occur. The prognosis as to complete recovery is absolutely unfavorable, death usually occurring as the result of septicemia, involvement of the medulla, some intercurrent disease, cystitis, bed-sores, etc.

Treatment.—No specific for this disease is known. The iodids may be given. The general health of the patient should be guarded. Local conditions, such as ulcerations, infections, etc., should receive appropriate attention, and the patient should be advised to avoid the risk of injury and to exercise great care in the treatment of what to him appear to be trivial injuries. Overexertion should be avoided.

LATERAL SCLEROSIS

This is a very rare disease, characterized by a primary degeneration of the corticospinal pathways.

Etiology.—The cause of this disease is obscure; overexertion, trauma, infectious diseases, etc., have all been named as causative factors. The disease usually occurs between the twentieth and the fortieth year, and in some cases more than one member of the same family may be affected.

Pathology.—Examination of the spinal cord of individuals who have been affected with this disease shows a degeneration which is practically limited to the pyramidal tracts, and which is always more marked in the lower regions of the cord than above.

Symptoms.—At the onset of the disease the patient first notices that he becomes fatigued very easily, and that there is a feeling of stiffness in the legs. The stiffness and the weakness gradually progress until a spastic paraplegia develops. The deep reflexes are markedly increased; the adductor reflexes are usually present. The Babinski sign, as a rule, appears early. The muscles retain their normal size, and there is no qualitative disturbance in the electric reaction. The power of walking is usually retained until late in the disease, when the gait becomes gradually more and more spastic. The muscular tension generally affects the extensors more than the flexors. Pain is slight or absent, and is due merely to muscular tension. There are no sensory disturbances, and the functions of the bowels and bladder are unaffected. As the disease progresses the upper extremities also become involved.

Diagnosis.—This disease is very uncommon. The symptoms of a spastic paraplegia are, in the great majority of cases, merely the early symptoms of some more diffuse disease; it is, therefore, highly important to bear this fact in mind, and to endeavor to discover the underlying condition. The disease must be differentiated from compression of the cord as the result of tumors, affections of the meninges and of the spine, myelitis, disseminated sclerosis, amyotrophic lateral sclerosis, hysteria, diseases of the brain that affect the motor tracts, such as tumors, vascular diseases, and the like, and from Erb's syphilitic spastic spinal

paralysis. From all these diseases lateral sclerosis is differentiated by the fact that it is very slowly progressive, and that there is a complete absence of any other symptom than spastic paralysis. There are no cerebral symptoms, no sensory symptoms, and no bowel or bladder involvement.

Course and Prognosis.—The disease is a slowly progressive one, extending over many years. The prognosis as to recovery is unfavorable, but as to life and usefulness it is good.

Treatment.—Overexertion should be avoided and trauma guarded against. The general health should be maintained by proper diet, fresh air and tonics, and a moderate amount of exercise is to be advised. The spastic condition of the muscles and the tendency to spasmodic contractions of the muscles is best relieved by warm baths, hot applications to the spine, massage of the affected parts, and the administration of sedatives.

ATAXIC PARAPLEGIA

This is a chronic disease of the spinal cord characterized by symptoms that can be referred to the disturbance of functions of the posterior and lateral tracts of the cord.

Etiology.—This disease usually develops in males after the thirtieth year. Trauma, exposure, and overexertion frequently precede the disease. In individuals suffering from this disease a marked anemia is usually present. Oppenheim believes that the anemia and loss of strength are not the cause of the disease, but that the anemia and the disease are both the result of some common cause, which may be a toxin or a micro-organism.

Marie traces this disease to an endarteritis in the spinal vessels.

Pathology.—Cross-section of the spinal cord of an individual who has had ataxic paraplegia shows degeneration of the column of Goll, part of the column of Burdach, the direct cerebellar tract, and the lateral pyramidal tract. The extent of the involvement of these different portions of the spinal cord varies in different cases—in some cases the posterior columns are affected more than the lateral, and in others the involvement of the lateral columns is more marked than that of the posterior

columns. In the majority of cases the dorsal region is first involved and shows the most extensive lesions.

Symptoms.—Two groups of symptoms characterize this disease, either of which may be the first to appear, and either of which may merge into the other, depending upon whether the posterior columns or the lateral columns show the greater involvement, and upon the extension of the disease as it advances. In the first group the symptoms are spastic—spinal paralysis, accompanied by ataxia, lightning pains, bladder weakness, and slight sensory disturbances, such as loss of muscle sense and incoördination. In these cases the disease begins with a feeling of fatigue after mild exertion, numbness, heaviness, and stiffness in the legs, with unsteadiness of gait, which is usually worse in the dark. There is generally decrease of tactile sensibility in the feet, an exaggeration of the knee-jerks, and the Babinski sign may be present.

In the second group the symptoms are of the tabetic type. The disease begins with ataxia, lightning pains, and bowel and bladder disturbances, to which is added a progressive paraplegia. In this group of cases the onset is similar to that of tabes dorsalis, except that it is somewhat more rapid, and that the symptoms of a paraplegia are added.

As the disease advances either the spastic or the tabetic symptoms may become more prominent, the spastic usually predominating over the ataxic symptoms. The case presents an appearance similar to that of lateral sclerosis to which sensory disturbances are added.

Diagnosis.—This disease must be differentiated from multiple sclerosis and cerebrospinal syphilis. From *multiple sclerosis* it is differentiated by the absence of speech disturbances, nystagmus, intention tremor, etc. From *spinal syphilis* it is differentiated by the examination of the cerebrospinal fluid, the presence of cerebral symptoms, local pain and stiffness of the back, and the more rapid progress of symptoms, with exacerbations at night.

Course and Prognosis.—The course of this disease is usually chronic or subacute; it may run its course within a few months or it may last many years. It has a tendency to spread from below

upward, but may affect all four extremities simultaneously. The prognosis is grave, but not hopeless, for some of the cases that develop as the result of, or during the course of, anemia may recover.

Treatment.—In those cases in which marked anemia exists the treatment should be directed toward the relief of this condition. Proper food, arsenic and iron, fresh air, attention to the gastro-intestinal tract, careful examination to discover the cause of the anemia and to correct it if found make up the treatment. Those cases in which examination does not reveal a cause are to be treated by making the patient as comfortable as possible by means of warm baths, relief of pain, massage, and attention to the general health.

FRIEDREICH'S DISEASE (HEREDITARY ATAXIA)

This disease is characterized by ataxia developing in childhood, and by the fact that more than one member of the same family usually become affected.

Etiology.—This disease is due to defective development of the spinal cord. Direct heredity is unusual, but a history of epilepsy, mental disease, alcoholism, etc., in the ancestry is generally present. In some families one member may be affected with Friedreich's ataxia; brothers or sisters have either the same disease or suffer from epilepsy or mental abnormalities. The disease may manifest itself first after an infectious disease, injury, or malnutrition; in some cases the condition may be congenital. As a rule, the symptoms make their appearance between the sixth and the eighth year, but sometimes they do not appear until puberty.

Pathology.—The spinal cord as a whole is thinner than normal, and cross-section will usually show degeneration of the columns of Goll and Burdach, of the direct cerebellar tract, of Clark's column, and of the pyramidal tracts. As a rule, some fibers in these tracts are spared. Wherever nerve-fibers are wanting, a thickening of the glia tissue is evident.

All investigators agree that this disease is due to an arrest of development of the various systems of fibers in the spinal

cord, being most marked in the posterior portion of the cord.

Symptoms.—The affected child, as a rule, shows a disturbance of gait; this is uncertain and staggering, and because of this

Fig. 121.—Friedreich's ataxia.

unsteadiness the patient walks with the legs wide apart (Fig. 121). If the disease advances this ataxic condition becomes apparent in the attempt to maintain any position, such as sitting or standing; there is a more or less constant swaying of the body (static ataxia). The ataxia spreads slowly, and sooner

or later the upper extremities become affected. Choreic movements sometimes occur, and in these patients involuntary purposeless movements are made from time to time, the various muscles of the body being affected. Muscular weakness may be associated with ataxia, but it is only in the last stages of the

Fig. 122.—Friedreich's ataxia, showing lateral curvature of spine.

disease that weakness becomes marked; it then may be combined with contractures and atrophy. As the ataxia increases a certain rigidity of the legs begins to appear; this is commonly attended by contracture of the posterior tibial group of muscles, causing a talipes equinus.

There is also a hyperextension of the toes, especially of the basal phalanx of the great toe, while the terminal phalanx is in flexed position, giving rise to the condition known as hammer-toe. As the disease progresses scoliosis frequently develops, and eventually walking or standing alone becomes impossible (Fig. 122). The tendon reflexes usually disappear early in the disease, although there are exceptions to this rule. Babinski's sign can sometimes be elicited. Sensory disturbances are not prominent, although there may be some decrease in the sense of touch and in the sense of position. Bowel and bladder disturbances are very uncommon. The speech of these patients is usually slow, indistinct, poorly articulated, and difficult to understand. In some cases, particularly after the disease has lasted for some time, the speech becomes very irregular—some words and syllables are brought forth slowly, whereas others are enunciated rapidly, resulting in jerky speech. The patient usually presents a somewhat stupid appearance, and many are incapable of intellectual development. Early in the disease nystagmus develops, which is not apparent except on attempts to rotate the eyes from side to side. Paralysis of the ocular muscles does not occur, and there is no disturbance of the pupillary reflexes.

Diagnosis.—This disease must be differentiated from *multiple sclerosis:* multiple sclerosis does not begin with ataxia, but if this is present, it is generally associated with a spastic paralysis and with increase of tendon reflexes; affections of the ocular muscles are common and are absent in Friedreich's ataxia. The abnormal movements in multiple sclerosis are of an intentional character, and in Friedreich's ataxia they are of a choreic nature. In multiple sclerosis there are no abnormal movements while the patient is at rest, and in Friedreich's ataxia static ataxia is usually present.

From *hereditary syphilis* this disease is differentiated by the fact that hereditary syphilis is usually of an acute or subacute onset; there are marked remissions in the symptoms, and the optic and oculomotor nerves are frequently affected. Examination of the blood and spinal fluid will clear up the diagnosis.

From *locomotor ataxia* this disease is differentiated by the

age of onset, the frequency of bowel and bladder disturbance, pupillary disturbances, lightning pains, and absence of contractures, nystagmus, and speech disorders.

From *multiple neuritis* it is differentiated by the mode of onset, the presence of paralysis and of pain, the history of the case, the absence of speech disturbances, nystagmus, etc.

From *Marie's cerebellar hereditary ataxia* it may not be possible definitely to differentiate Friedreich's ataxia. The distinguishing facts are that Marie's cerebellar ataxia usually develops between the twentieth and the thirtieth year; it affects the arms before the legs; the characteristic deformity of the feet is not present, whereas pupillary disturbances and optic atrophy are frequently observed.

Course and Prognosis.—This disease usually begins in early childhood, runs a very slow course, and may last for many years. As a rule, from ten to fifteen years elapse after the onset of the disease before the patient becomes incapable of getting about without assistance; after this, if the patient is well cared for, he may live for many years with a fair degree of comfort. The prognosis is very unfavorable, as no form of treatment seems able to arrest the progress of the disease.

Treatment.—This is confined to looking after the patient's general health and to maintaining his nourishment as well as possible.

Marie's Cerebellar Hereditary Ataxia.—This disease is usually described along with Friedreich's ataxia. It is due to arrest of developement of the cerebellum. It usually manifests itself about the age of twenty years. The tendon reflexes are not disturbed. As a rule, oculomotor paralysis, especially ptosis and abducens palsy, immobility of the pupils, optic atrophy, and cerebellar ataxia are present. In some cases intention tremor also occurs.

Diagnosis of this disease from Friedreich's ataxia is not always possible, and many authorities believe this to be merely one of the types of hereditary ataxia in which there is a prominence of bulbar and cerebellar symptoms. The course, prognosis, and treatment are similar to those of Friedreich's ataxia.

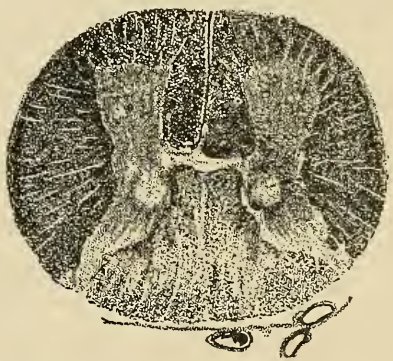
LOCOMOTOR ATAXIA (TABES DORSALIS)

Locomotor ataxia is one of the commonest diseases affecting the spinal cord.

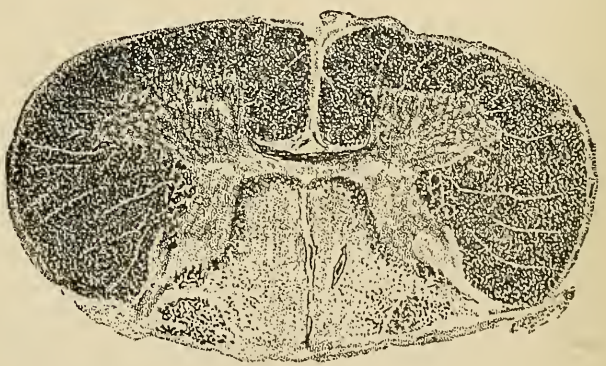
Etiology.—Chronic intoxications, injuries, infectious diseases, overwork, exposure to cold, etc., are probably among the predisposing causes of this disease. Syphilis is certainly the causative agent in practically all cases. There are a few cases, however, in which the most careful examination fails to reveal a preëxisting syphilitic infection. In the majority of instances locomotor ataxia does not manifest itself until within from five to fifteen years after the infection, and it is also usually observed that in these cases the symptoms of the syphilitic infection have not been very severe.

Pathology.—Examination of the nervous system of an individual who has been affected with a moderately severe form of locomotor ataxia will show pathologic changes in the spinal cord, posterior roots, posterior root ganglia, spinal meninges, cranial nerves, and, to more or less extent, the peripheral nerves. Degeneration of the posterior columns of the spinal cord is present, varying in extent, and being usually more marked in the neighborhood of the posterior horns (Fig. 123). There are always some fibers in the posterior columns that escape this degeneration. Many fibers in the posterior horn, especially those ending around cells in the column of Clark, also show degenerative changes. The posterior roots are usually smaller than normal, and contain atrophied fibers. In the ganglia of the posterior roots many cells are seen in different stages of degeneration, with consequent degeneration of their processes, and similar degenerative changes are also seen in the ganglia of the sympathetic system. Occasionally examination of the peripheral nerves also discloses degenerated fibers, but these changes are not so constant as those occurring in the spinal cord. Along with the process of degeneration of the fibers in the cord there is an increase of neuroglia, with subsequent sclerosis, bringing about a flattening of the posterior portion of the cord. The meninges of the cord also undergo thickening, and there is a closer adhesion between the meninges and the cord than is

Dorsal region showing degeneration in Goll's tract in the early stage of locomotor ataxia (Oppenheim).



Dorsal region showing marked degeneration of Goll and Burdach and Clark's column in a well-advanced case of locomotor ataxia (Oppenheim).



Cervical region showing marked degeneration of the posterior columns in the terminal stage of a case of locomotor ataxia (Oppenheim).

Fig. 123.—Cross-sections of spinal cord in locomotor ataxia, stained by Weigert-Pal method, showing degenerated tracts light in color.

present in normal conditions. When this affection extends to the medulla, there is usually involvement of the Gasserian ganglion, proximal root of the fifth nerve, and its prolongation into the medulla and spinal cord. Occasionally the ninth and tenth nerves are involved. In about 10 per cent. of cases there is primary degeneration of the optic nerve. The oculomotor nerves may be involved in some cases, although their nuclei seem to undergo no change. There are two theories to account for the location of the lesion in this disease: the first holds that, as the result of the action of some toxic substance, the cells in the ganglion on the posterior roots, the ganglion on the cranial nerves, or the ganglion of the sympathetic system, undergo varying degrees of degeneration, and as a consequence their processes, both those which enter the spinal cord and to a less extent those which go to the peripheral nerves, undergo degenerative changes. No satisfactory explanation has been given why those processes that help form the peripheral nerves are less affected than those that enter the central nervous system. The second theory maintains that pressure is exerted upon the posterior roots at the point of entrance into the central nervous system by a thickening, with subsequent contraction, of the meninges, which has resulted from the effects of prolonged irritation by some toxic substance; as a result of this pressure on the posterior roots atrophy occurs, with degeneration of the fibers entering the cord. This theory does not hold in view of the fact that such a thickening of the meninges does not exist in all cases of locomotor ataxia.

Symptoms.—The symptoms of locomotor ataxia are very numerous. Nevertheless certain symptoms occur more frequently than others, and whereas some are prone to occur early in the disease, others manifest themselves more frequently in the later period of the affection.

For descriptive purposes, the disease is usually considered as divided into three stages: the *initial stage*, in which most of the subjective symptoms are of an irritative character; the *second stage*, in which there may be an accentuation or a disappearance of some of the symptoms of the first stage, together with marked disturbances of coördination; the *third* or *terminal*

stage, which is characterized by general physical disintegration and progressive loss of motor power.

First Stage.—Among the early symptoms, paresthesias are the most common, the patient complaining of tingling, numbness, or formication in the lower extremities, or of a feeling as if there were something soft under the feet when he walks. Unusual sensations of heat or cold in various parts of the body are felt, and there is generally undue fatigue upon slight exertion. These paresthesias are usually felt first in the legs, and later on in the trunk; in some cases the upper extremities are first affected, and there is at times a sensation of constriction, as if a band or cord were tied about the body. This is known as the girdle sensation. All these abnormal sensations may be constant or intermittent in character, and may continue throughout the disease or disappear during any stage.

Pain.—This may be mild or extreme in character, and sometimes precedes other symptoms for a considerable period. The pain is of a sharp, shooting, burning character, intermittent and paroxysmal in occurrence. These pains have been termed "lightning pains." The pain does not follow the course of the nerves, but is usually said to be felt deep in the tissues; it is frequently treated as rheumatic before the true nature of the disease is recognized. The attacks of pain are particularly prone to develop in cold, damp weather, may continue throughout the disease or disappear at any stage.

Crises.—These attacks are probably due to irritation of the nerves or sympathetic fibers which are supplied to the affected organs, and are similar in origin to the paresthesias and painful attacks which affect the limbs and trunk. The *gastric crises* are probably the more common variety. In this condition there is sudden severe pain in the stomach, accompanied by nausea and vomiting, which may continue for hours or days. This form of gastric disturbance is very intractable, for the stomach seems unable to retain anything. At times the attacks of pain and vomiting are followed by shooting pains in the arms or lower extremities. The attacks terminate suddenly, and are followed by marked weakness, loss of weight, and considerable mental distress.

Intestinal or *rectal crises* may accompany the gastric crises. These are usually ushered in with severe pains in the intestines and rectum, accompanied by marked tenesmus. These intestinal disturbances are less common than the gastric crises, and are more apt to occur later in the disease.

Laryngeal Crises.—These are marked by sudden severe persistent coughing and difficulty in breathing. The cough is loud and harsh, and is more or less constant. In addition to the great difficulty in breathing there is increased frequency of respiration. The voice becomes hoarse and rough. In *pharyngeal crises* there occur rapid movements of swallowing, which are painful in character and are followed by noisy belching. Painful disturbances of the functions of other organs of the body have also been described.

A thorough and careful examination of patients affected with these different forms of crises fails to reveal a cause for their occurrence, and they have been said to be due to a central irritation of the sensory nerves supplying the affected parts.

Disturbances of bowel and bladder control are also commonly complained of early in the disease. In some cases the bladder or the rectum empties itself without the knowledge of the patient, the first intimation of the involuntary movement being had when he feels the urine or the feces on the skin. In other cases there is a retention of urine, the patient being unconscious of the fact that the bladder is unusually full, or that it may never completely empty itself. Marked constipation may occur, and it may be impossible to empty the rectum without artificial assistance. Increased or decreased or loss of the sexual power may occur in the early stages. In the later stages there is usually complete impotence.

Physical Signs.—In the first stage the most common physical signs are Westphal's sign, or loss of the knee-reflex; Argyll Robertson's sign, that is, reflex immobility of the pupils to light, the accommodation reflex being retained; areas of decrease or loss of tactile sensibility on the trunk. Less commonly there occur transient oculomotor paralysis, optic atrophy, trophic disturbances, deafness, hypotonia of the muscles, loss or decrease of the sensation to pain or tactile sense, or of both, in the lower

extremities, loss of muscle sense in the lower extremities, Romberg's sign, etc.

Westphal's Sign.—This may appear years before other symptoms manifest themselves, and is one of the most frequent signs occurring in the initial stage. Jendrassik's maneuver should always be applied before determining definitely that the knee-reflex is lost. The tendo Achillis reflex is also generally absent. The reflexes in the upper extremities are generally preserved, and are often increased. In some cases they disappear before those of the lower extremities.

Argyll Robertson's Sign.—This sign is present as a very early symptom in about 90 per cent. of the cases of locomotor ataxia. It consists of a loss of pupillary contraction upon exposure to light, with retention of pupillary contraction to accommodation. In testing for this sign it is always well to have the patient look at a distant object, and then alternately expose the eye of illumination and shield it against it. Under normal conditions upon exposure to light the pupils will contract: when the Argyll Robertson's sign is present, there is no contraction upon exposure to light. By having the patient look at a distant object the pupillary contraction that occurs during the act of accommodation is eliminated.

Objective Sensory Disturbances.—Frequently there is observed early in the disease the existence of small areas of loss of tactile sense upon the trunk (Fig. 124). These are seen especially on the back, beneath the shoulder-blades, and in the lumbar region. These areas may exist even when there is no loss of sensation in the lower extremities. Decreased or delayed transmission of pain, temperature, or tactile sense, or all of them, and loss of the muscle sense in the lower extremities, always occur at some stage of the disease—usually toward the end of the first stage. As a result of the disturbance of muscle sense there is a peculiarity of gait, known as the ataxic gait. This peculiarity in walking appears at some period in almost every case, and from this the disease has derived its name—locomotor ataxia. The loss of pain sense is usually found in the lower extremities, in irregular areas, and may occur even though tactile sensibility is intact. In other cases in conjunction with the disturbance of pain sense

there is also a loss of tactile sense. In the great majority of cases disturbance of muscle sense is present; this is especially so in the lower extremities, the patient being unable to distinguish, with his eyes closed, the various states of flexion or extension of the great toes. As the disease advances this disturbance is present in the ankle-joint, knee-joint, hip-joint, and upper extremities.

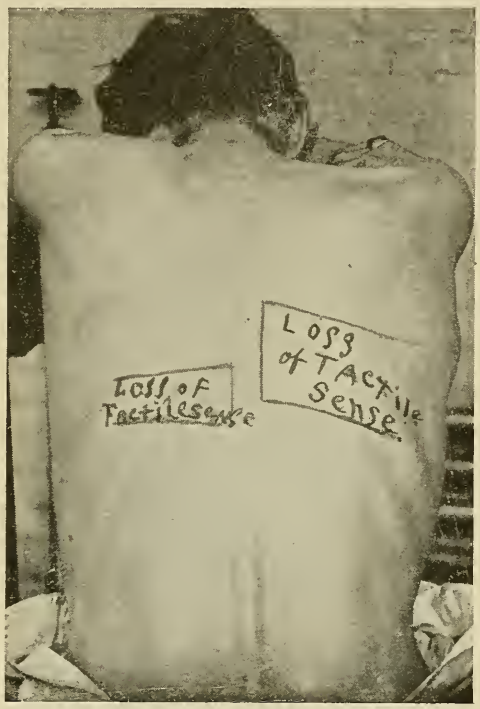


Fig. 124.—Tactile disturbance upon trunk, frequently seen early in disease.

Romberg's Sign.—This consists of swaying of the body when the eyes are closed and the feet are placed close together. This symptom may appear early, but is more commonly present in the later period of the initial stage. As the disease progresses the swaying occurs even when the eyes are open and the feet are far apart.

Muscle Hypotonia.—In this condition there are flaccidity and relaxation of the muscles, so that the limbs can easily be overextended or overflexed. This manifestation is more apt to

occur in the lower extremities, producing genu recurvatum; it may also occur in other joints of the lower extremities and in the upper extremities or trunk, allowing undue movements of these parts to take place. This symptom usually occurs in the second stage of the disease.

Disturbances of the functions of the cranial nerves may also be among the early or late symptoms; the most frequent of these is an oculomotor paralysis. This is usually a temporary condition. A permanent oculomotor paralysis is, as a rule, ob-

Fig. 125.—Charcot joints, both knees.

served only in the later stages of the disease. Oculomotor paralysis may manifest itself in a ptosis; as paralysis of the external rectus muscle; sometimes in one or more branches of the third nerve, or complete ophthalmoplegia may occur. The paralysis of the ocular muscles is usually irregular, and may involve one or more muscles of one or both eyes. The pupils may be abnormally small, or there may be an inequality in their size.

In about 10 per cent. of the cases a bilateral simple progressive

atrophy of the optic nerves is among the earliest symptoms, and usually leads to complete blindness. This may, for a long time, be the only evidence of tabes, and it is only in exceptional cases that it develops during the late stage of the disease. The fifth nerve may also be affected in this disease, and when this occurs there may be attacks of pain, a feeling of numbness in the face, with disturbance of sensation, particularly decreased

Fig. 126.—Charcot joints, one knee.

perception of painful stimuli in the distribution of the fifth nerve. The symptoms referable to lesions of the ninth and tenth nerves are marked increase in the pulse-rate, which is found in many cases of tabes, often in the early period, and cardiac crises, gastric crises, laryngeal crises, etc.

Trophic Disturbances.—Trophic disturbances in the joints and bones—tabetic arthropathies—usually appear early in the dis-

ease, and are not uncommon. The knee-joint, either of one or both sides, is that most frequently affected. This disturbance may be atrophic or hypertrophic in nature, and usually begins suddenly. There is swelling of the joint, which also involves the surrounding tissue, and an exudate into the joint. There is no pain, redness, or fever. In the atrophic variety there are more or less complete destruction of the cartilage of the joint and extreme mobility, together with luxation or subluxation of the

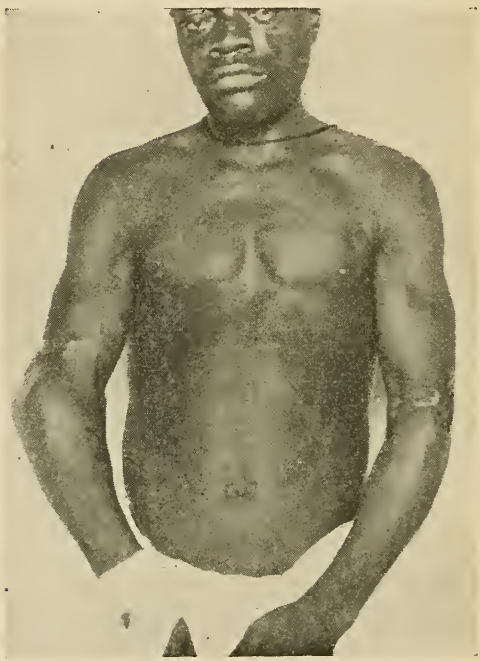


Fig. 127.—Charcot joints, one elbow.

bones. In the hypertrophic form there is a formation of new bony tissue, with bony excrescences in and about the joint (Figs. 125–127).

The pathologic processes that take place are closely related to those of arthritis deformans, but may be distinguished from them by the absolute freedom from pain. In some instances there is an extensive effusion into the joint, this being rapidly reabsorbed, without destruction of the articular ends of the bones or formation of new tissue taking place.

The explanation offered for the occurrence of these arthropathies is that they are the result of trauma or strain, which, owing to the anesthetic condition of the joint, is not perceived, and consequently no effort is made by the individual or by nature to shield or spare the affected joint. Furthermore, there being an interference with the conduction of afferent stimuli from the joint to the trophic center of the cord, no well-organized effort is made on the part of nature to repair the damage that has been done, and consequently the reparative processes which take place are irregular, excessive, or insufficient.

In some cases the bones are abnormally brittle, so that fractures occur spontaneously. Perforating ulcers develop quite commonly, usually on the sole of the foot, in the region of the balls of the toes; these show a great tendency to penetrate deeply, and are very resistant to treatment. The skin may also be the seat of trophic disturbances; thus there may be herpetic eruptions, thickening of the skin, or an imperfect growth or falling out of the nails. Subcutaneous ecchymoses may be present, and are usually secondary to trauma that has not been perceived.

These symptoms and signs may or may not appear in the early stage of the disease, or they may appear and disappear in part, or become accentuated as the disease enters on the second stage, or this may set in without any of these symptoms or signs having been observed. The first evidence of the disease may be the appearance of the symptom most characteristic of the SECOND STAGE—namely, ataxia.

Ataxia may be defined as an imperfect coördination of muscular action or awkwardness of movement. This incoördination, in the majority of cases, first affects the muscles of the lower extremities, and becomes exaggerated when the control of the eyes is excluded; consequently it is more marked while walking or moving about in the dark and in going down steps. Ataxia in the lower limbs produces the characteristic gait of tabes. In this condition the steps are irregular in length; the patient walks with feet wide apart, and the muscular contractions are excessive. The legs and feet are raised too high from the ground, and the foot is brought to the ground with excessive force, the

heel striking first. As the disease progresses ataxia affects all the movements of the body. When the patient rises from a sitting position he is apt to lean too far forward, and upon assuming the erect position he sways backward and forward before gaining his proper balance.

These patients usually watch all their voluntary movements carefully, as they are performed better with the aid of sight than by depending upon the disturbed coördination. The hands and upper extremities may also become ataxic, when the same peculiarities and awkwardness are apparent in the movements of the upper extremities. When ataxia is well developed, there is commonly a marked disturbance of the deeper forms of sensibility, such as the pressure sense, the sense whereby the difference in weights is perceived, the sense of position, etc.

In the second stage there is an intensification of the signs of the first stage. There may occur an accentuation, a decrease, or a disappearance of some of the symptoms. In this stage there is nearly always a loss of sensibility. Areas of anesthesia are present that correspond to the distribution of the spinal-cord segments and peripheral nerves. There may be a loss of or a marked delay in the transmission of the various forms of sensation.

Pain and temperature sense are particularly apt to be disturbed. The muscular strength of the patient is usually good in this stage, although lack of coördination may give rise to a semblance of weakness.

Third Stage of Paralysis.—The second gradually merges into the third stage. When the ataxia becomes so marked, the patient is unable to leave his chair or bed. In this stage the signs and symptoms that have appeared in the two previous stages usually become exaggerated in intensity. As a result of bowel and bladder disturbances, crises, attacks of pain, etc., there is marked interference with the general nutrition of the patient, and complications secondary to infection of the bladder or kidneys, etc., may appear. The patient usually dies of some intercurrent disease. The paralysis is due principally to the marked ataxia and to the general disturbance of nutrition. If the disease is not arrested, unusual symptoms may appear.

Among these are mental disturbances, attacks of hemiplegia, epileptiform seizures, involuntary movements, etc.

Course.—The disease usually pursues a very chronic course. In most cases the onset is marked by symptoms of irritation, such as pain, paresthesia, crises, etc. A few cases begin with progressive loss of vision, and in a few the first evidence of the disease is the ataxia. The disease generally runs a very prolonged course, some symptoms disappearing and others appearing as the disease advances.

An individual may have tabes for many years without being conscious of the fact. The disease usually lasts from fifteen to thirty or forty years if the individual does not die of an intercurrent disease or of some complication.

Prognosis.—The prognosis as to life is good except in those cases subject to severe crises, during any one of which the patient may die of exhaustion. There is also danger to life from infection of the bladder and kidneys, from sepsis the result of bed-sores, or from injuries that are not painful, etc. The prognosis as to arrest of the disease and eradication of the underlying syphilitic infection is, in my opinion, very good if the proper treatment is instituted. One must remember that, although it may be possible to prevent the further progress of the disease, it is not possible to restore to the normal those tissues that have been destroyed; consequently many symptoms may be permanent. This last is especially true of paresthesia, anesthesia, painful attacks, the various physical signs, such as pupillary disturbances, loss of the knee reflexes, etc. The ataxia can often be overcome by a course of muscular "reëducation."

Diagnosis.—The diagnosis of tabes, as a rule, presents no difficulty. The principal disease from which it must be differentiated is multiple neuritis, the differentiation being made from the history of the onset: In multiple neuritis the onset is usually acute or subacute in nature, the disease reaching its height in a few weeks or months. In tabes the onset is much more gradual. In multiple neuritis sensory disturbance, manifesting itself in glove-shaped or stocking-shaped areas, or along the distribution of the peripheral nerves, occurs; in tabes the sensory

symptoms affect the distribution of the spinal segments. In multiple neuritis there is an absence of primary optic atrophy, Argyll Robertson pupil, bladder disturbances, and girdle sensation, whereas one or more of these symptoms are practically always present in tabes. In multiple neuritis the peripheral nerves are tender to pressure and there is usually a degenerative paralysis; in tabes the nerves are not tender to pressure, and paralysis rarely occurs. In multiple neuritis there is generally a history of toxemia, whereas in tabes syphilis is the basic factor.

In tabes it is extremely important that the diagnosis be made early, although it is only in the early stage that there is any difficulty in making the diagnosis. The existence of any one of the following group of symptoms justifies a diagnosis of tabes. In the absence of mental symptoms an early diagnosis can be made when examination of the cerebrospinal fluid gives a positive Wassermann reaction, and this is combined with any two of the following signs: Argyll Robertson pupil, Westphal's sign, optic atrophy, crises, Charcot joints, bladder disturbances, etc.

Argyll Robertson pupil, a positive Wassermann reaction of the cerebrospinal fluid, or Westphal's sign, in conjunction with any two of the common symptoms of tabes, practically justifies a diagnosis of tabes.

The cerebrospinal fluid in at least 95 per cent. of cases of tabes gives a positive Wassermann reaction, and in addition to this there is a marked lymphocytosis. In all cases in which there is any doubt as to the diagnosis, examination of the cerebrospinal fluid is of the greatest assistance.

Treatment.—The treatment resolves itself into the relief of such symptoms as may exist, and in efforts directed toward the arrest of the disease. Patients seem to do better in dry, warm climates than in moist, cold climates. As a rule, these patients are hypersensitive to cold, and they should, therefore, be given only warm or tepid baths. The maintenance of the general nutrition of the patient is most important, and especial care should be directed toward this point. As these patients tire very easily, and as fatigue is injurious, they should be warned against overexertion. If there is anesthesia in any areas of the

body, the patients should be warned against exposing themselves to trauma, and all trifling injuries should be carefully and promptly looked after, thus avoiding the development of more serious conditions. Proper attention should be directed toward the bowel and bladder functions, and care should be exercised that the bladder does not become overdistended. When catheterization is necessary, it should be done under aseptic precautions. For relief of the pain many drugs have been used; among these are phenacetin, pyramidon, aspirin, and codein, and eventually, in most cases, morphin will be resorted to. For the various crises morphin and atropin in large doses and counterirritation along the spinal column seem to be of value. Continuous galvanism to the spine and to the cervical sympathetic ganglia has been recommended.

Suspension, massage, nerve-stretching, etc., have all been used, with little or no benefit. Apparatus for supporting the spinal column has no special value, but where deformity or hypotonia exists, apparatus for the support of the legs is recommended. Sexual excesses are to be avoided. When ataxia is present, or early in the disease, if there is awkwardness of movement, a system of muscular exercises recommended by Frenkel, of Hayden, is of great value. These exercises are very simple, and when properly carried out, patients suffering even with extreme ataxia can be reëducated to use their extremities in a normal manner. In severe attacks of crises, when other means have failed, relief may be obtained by the injection into the subdural spinal space of a small quantity of a 25 per cent. solution of magnesium sulphate.

Föerster and Frenkel have both described a surgical procedure for the cure of gastric crises. These operations are attended with considerable risk and are not always successful.

In view of the results that have been attained in the past two years in the treatment of tabes by intraspinous medication, there can be no question as to the advisability of employing this form of treatment. Before beginning the treatment a Wassermann examination of the blood and cerebrospinal fluid, and a cell count, globulin reaction, etc., of the spinal fluid should be made. In those patients in whom a positive Wassermann reaction of the

spinal fluid is obtained the following line of treatment is advisable:

The patient is given an intravenous injection of $\frac{6}{10}$ gram of salvarsan. Within one hour 80 c.c. of blood are withdrawn, under the strictest aseptic conditions. The serum is separated from the cellular constituents of the blood either by centrifuging or by allowing the blood to stand for about twenty-four hours. The serum is now diluted with normal salt solution until a 60 per cent. solution is obtained; to each 30 c.c. of this solution there is added $\frac{1}{50}$ grain of mercuric chlorid. The serum is now heated to 56° C. for half an hour. A spinal puncture is made, and from 20 to 40 c.c. of cerebrospinal fluid are withdrawn, and then, under aseptic precautions, and very slowly, the serum is introduced into the subdural space. This treatment is repeated at intervals of from three to four weeks, until the cerebrospinal fluid gives a negative Wassermann reaction, a normal cell count, etc. In those cases in which the blood test is positive, mercurial inunctions daily and potassium iodid in increasing doses in the intervals between the intraspinous injections are indicated. A Wassermann examination and a cell count of the cerebrospinal fluid withdrawn should be made before the intraspinous injection, so that the effects of the treatment may be observed. When the spinal fluid yields a negative Wassermann reaction and a normal cell count, it is safe to assume that the syphilitic infection of the nervous system has been eradicated. It is advisable, nevertheless, to repeat this examination at intervals of six months for the following two years in order to be certain that the disease has been cured. As a result of these injections there is usually a mild reaction, manifested by a slight rise of temperature, a feeling of general malaise, diarrhea or vomiting, pain in the legs, and sometimes severe headache. This reaction rarely lasts more than twenty-four hours. In quite a number of cases this method of treatment has been followed by marked improvement in the patient's general condition: the lightning pains, bowel and bladder disorders, disturbances of gait, etc., have disappeared. This treatment does not, however, restore to functional capacity that portion of the spinal cord that has been destroyed as the result of the disease.

Those symptoms that are due to irritation are relieved, and it would appear that the treatment arrests any tendency for the disease to progress.

PRESSURE ON THE SPINAL CORD

The spinal cord may be compressed as the result of the following conditions: spinal caries; malignant growths of the vertebræ or meninges; syphilitic exudations into the spinal canal; aneurysms of the aorta, causing absorption of the vertebral bodies, etc.

Spinal Caries.—*Etiology.*—Tuberculosis and injury are the usual causes of spinal caries. Children are affected more frequently than adults, and any portion of the spinal column may be involved.

Pathology.—The condition is almost always tubercular in nature, but may occur after injury. It usually affects the bodies of the vertebræ first, less frequently the joints, ligaments, or vertebral arches and their processes. As a result of osteitis there is a breaking down and softening of the bone, with the formation of a caseous or purulent infiltrate. The process is generally limited to one vertebra. If cure takes place, new bone is formed at the site of the diseased process. If the disease progresses there are absorption and necrosis of the bony substances, with consequent collapse of the neighboring vertebræ, or the soft, caseous material forms an abscess that compresses the spinal cord. Rarely is the spinal cord compressed by the vertebra itself (Figs. 128, 129).

Compression of the cord may also occur without displacement of the vertebræ. Usually the narrowing of the spinal canal takes place slowly. If there is pressure against the dura, a more or less chronic inflammatory process occurs that extends beyond the confines of the diseased vertebræ, bringing about marked increase of the normal thickness of the dura, with some bulging into the canal. This process is generally slow in advancing. The compression and obstruction of the blood and lymph-vessels in the membranes and cord produce edema of the cord, which may exist for a prolonged period with-

out producing any marked alteration in the nerve elements.

Fig. 128.—*x*-Ray of spinal column in spinal caries.

Sooner or later, if the pressure is continued, destruction of the nerve elements follows. Softening and inflammation bring

about a more or less well-marked transverse myelitis. In some instances there is direct evidence of the tubercular infection of the spinal cord. This is particularly marked around the vessels. As a rule, the edema, softening, or myelitis involves several segments of the cord. If the disease is of long standing, signs of secondary degeneration are also present. The nerve-roots are affected as a general rule, being subject to pressure in the

Fig. 129.—Photograph showing extensive spinal curvature without involvement of cord. (Case of Dr. A. Shipley.)

spinal canal or in their passage through the intervertebral foramina.

Symptoms.—The general condition of the patient is not always affected. In some cases there may be constitutional symptoms and evidence of tuberculosis may exist in another portion of the body. Usually, the first and one of the most common symptoms is pain, which is felt in the region of the affected vertebræ, and is increased on movement or on pressure. The pain radiates along the distribution of the nerves whose trunks or roots are

involved in the pathologic process. The pain is generally very intense, and the patient endeavors to guard against all shocks and avoids any unnecessary movements.

There is rigidity of that portion of the back that is involved in the pathologic process. Deformity of the spinal column may exist, or the vertebral disease may evidence itself by abscess formation at some other point. As the result of pressure on the cord two sets of symptoms occur: 1st, The root symptoms, which consist of pain in the distribution of the affected roots; this pain may exist long before other symptoms appear. If the disease advances, there is added to the pain atrophic paralysis, with disturbance or loss of sensation in the portion of the body supplied by the affected nerves. 2d, As the result of pressure on the cord there is interference with the transmission upward and downward of stimuli. In addition to the segmental symptoms and those due to interference with the transmission of impulses in the cord, in severe cases there usually appear trophic disturbances, such as bed-sores, ulcerations, etc.

Diagnosis.—This, as a rule, presents no difficulty. It may be formulated in the absence of deformity by the presence of tenderness, spinal rigidity, the root and cord symptoms, and by means of the *x*-ray, which will demonstrate the presence of disease of the vertebræ. The condition must be differentiated from osteomyelitis vertebralis, which usually runs a much more acute course, with high temperature and severe constitutional disturbances, together, generally, with a history of similar affection in other bones or of some preëxisting focus of infection. Spinal caries must also be differentiated from spondylitis typhosa: in the latter condition the pains are of unusual severity, of more or less constant duration, and there is a history of typhoid fever; there is also a rise in temperature, and very few spinal cord symptoms are present.

Course.—The disease usually runs a very chronic course. In the majority of cases the symptoms of the tubercular bone condition may long precede the appearance of nervous symptoms. In some cases, particularly in adults, the roots and spinal cord symptoms may first give warning of the presence of the disease; in a few cases the disease manifests itself suddenly.

Prognosis.—The prognosis is largely dependent upon the stage at which the disease is treated, and upon the thoroughness with which the treatment is carried out, and also upon the general health of the patient. If the disease occurs in well-nourished individuals and is recognized fairly early, under proper treatment the prognosis is very favorable. If the general condition of the patient is good, many cases that have gone on to paralysis and loss of bowel and bladder control, if the sensory disturbances are not marked, may make an almost complete recovery. As a rule, the prognosis is fairly good if the patient's general condition is good.

Treatment.—It is most important to treat the patient's general condition. He should be given plenty of fresh air and good food. Climatic conditions constitute a factor, and general tonics should be administered. The treatment of the spinal condition consists in putting the parts at absolute rest, the application of plaster jackets, special apparatus, extension, etc. In those cases in which the pressure upon the cord is great, it is frequently advisable to apply extension apparatus for several weeks or months. The vertebral bodies must be relieved of all pressure and immobilized by the continuous use of plaster jackets or braces for from six months to two years after the disappearance of the nerve-pressure symptoms.

Malignant Growths of the Vertebræ.—When carcinoma appears, it is almost always secondary to carcinoma in some other part of the body, especially the breast. Sarcoma and osteosarcoma may originate in the vertebræ, but more frequently they arise in the vicinity and then invade the vertebral column. Other malignant growths may also either originate in the vertebral bodies or involve them secondarily.

These tumors, whether originating in the vertebræ or arising in the neighborhood and involving the vertebræ secondarily, extend more or less rapidly, finally causing either direct pressure upon the dura or spinal cord or, more commonly, eroding the dura and directly infiltrating both the membrane and the cord. These tumors may develop at any period of life, but usually occur in persons beyond middle life, and more frequently in women than in men. As a rule, the metastatic process is not

limited to the vertebral column, but affects other portions of the body as well.

Symptoms.—Root Symptoms: The earliest, and usually the most prominent symptom, is pain. This is always very severe, and is generally aggravated by movements or pressure. The pain is of an intense, burning, shooting character, its situation depending upon the location of the tumor. It may be unilateral at first, but soon becomes bilateral, and is frequently attended by hyperesthesia, muscular twitchings, and trophic disturbances, especially herpes. As the disease progresses evidence of spinal cord involvement is rapidly added to these symptoms.

Here again the symptoms are of two types, and depend for their distribution upon the particular portion of the cord involved. Segmental symptoms are due to the destruction of one or more segments of the cord, and consist of flaccid paralysis, loss of the reflexes, reaction of degeneration, vasomotor and trophic disturbances, and anesthesia. Tract symptoms, which are due to the interference of the transmission of impulses through the cord, consist of weakness or paralysis, loss of bowel and bladder control, increase of the deep reflexes, trophic disturbances, and disorders of sensation.

The symptoms are usually quite rapid in their advancement. The pain may be so intense as to give rise to a marked hysteric condition.

Diagnosis.—The diagnosis is based upon the patient's age, on the history or evidence of a primary growth elsewhere, on the marked pain in the vertebræ and in the distribution of one or two nerves, and on the deformity, if it should exist. *x*-Ray examination is sometimes of value, as are the comparative rapidity of the progress of the symptoms and the absence of tubercular disease.

The *prognosis* is absolutely unfavorable. The course is usually rapid, the duration being from a few months to a year. *Treatment* is confined to relieving the patient of pain and rendering him as comfortable as possible.

Aneurysm of the Aorta.—An aneurysm of the aorta may erode the vertebral bodies and gradually produce increasing pressure

upon the spinal cord. In those cases in which the evidence of an aneurysm of the aorta is present and the spinal symptoms occur subsequently the diagnosis is readily made. When pain of a neuralgic character, followed by symptoms of pressure upon the cord, or the symptoms of a superficial myelitis, are the first to appear, examination may reveal an unsuspected aneurysm of the aorta.

Symptoms.—The symptoms are those of a compression myelitis together with the physical signs and symptoms of aneurysm. In these cases the *x*-ray is of great value in the diagnosis.

Syphilitic Exudations into the Spinal Canal.—Syphilitic involvement of the vertebral bodies, while it may occur, is very rare. These syphilitic exostoses may attain considerable size and compress the nerve-roots or the cord; as a result of this pressure there may be pain in the distribution of the affected nerve and other pressure symptoms, giving rise to two types of symptoms. In addition, there may be large, irregular shaped exostoses which can be palpated in the back.

The *course* is usually slow, and recovery follows proper treatment. The *diagnosis* is made by careful physical examination and blood and spinal fluid tests, etc.

Treatment.—Rest, counterirritation, and antisyphilitic medication.

FRACTURE AND DISLOCATION OF THE SPINAL COLUMN

Dislocation occurs more frequently in the cervical region, and comparatively rarely involves the thoracic or lumbar region. The most important of the direct symptoms of dislocation is deformity of the spinal column; this is but rarely absent. The spinal column is kept rigid by the tension of the muscles, and every attempt at movement is painful.

Fractures of the vertebræ may occur in any part of the spinal column, and dislocation is frequently associated with the fracture. There is usually marked displacement, with deformity and muscular rigidity. In some cases the fracture may be present without any visible deformity. Occasionally in fracture or

dislocation the symptoms pointing to involvement of the nervous

Fig. 130.—*x*-Ray photograph of fracture dislocation of the spine.

system may be absent. As a rule, the spinal cord and nerve-roots are involved. In some cases the cord is compressed by

displacement of the vertebræ or by a fragment of bone that has become dislodged and has entered the spinal canal (Fig. 130).

There may be a stretching, crushing, or tearing of the cord as the result of excessive flexion or displacement of a portion of the spinal column. After such an injury there may be no visible evidence of the trauma inflicted on the spinal column. Compression of the spinal cord may also be caused by hemorrhage into the membranes of the cord or into the cord itself. It may also occur as the result of the excessive formation of callus in the processes of repair at the site of the fracture.

The spinal nerve-roots are either compressed close to the cord and along with it, or are crushed in their passage through the intervertebral foramina. The amount of involvement of the spinal cord varies—in some cases the cord may be extensively injured, and in others very slight injury is done. The symptoms depend upon the extent of injury to the cord, subsequent involvement of the cord by hemorrhage and inflammation, and also on the site of the lesion. In most cases the cord is affected at the level of the injury by compression, laceration, hemorrhage, and later by inflammation. Fracture and dislocation of the first two cervical vertebræ usually terminate fatally either immediately or within a short time. Fracture and dislocation of the third and fourth cervical vertebræ lead to involvement of the phrenic nerve; in these cases life is in great danger, fatal termination usually ensuing within a few days or weeks, being due to respiratory failure, etc. In fractures or dislocations below the fourth cervical vertebræ the symptoms pointing to disturbance of functions of segments of the cord and of the conduction of impulses through these segments usually clearly point to the area of the cord that is affected. In some cases the roots remain uninjured and the cord substance is involved: in these patients the symptoms will be situated lower down on the body than the point at which the injury occurred, and this difference in the level of injury and the level of the symptoms will be the greater the lower down in the vertebral column or cord the site of the injury is. In some cases there is a marked variation between the distribution of the symptoms and the site of the spinal injury: in these cases the cord is usually only

partially affected, and there frequently exists a central hemorrhage (Figs. 131, 132).

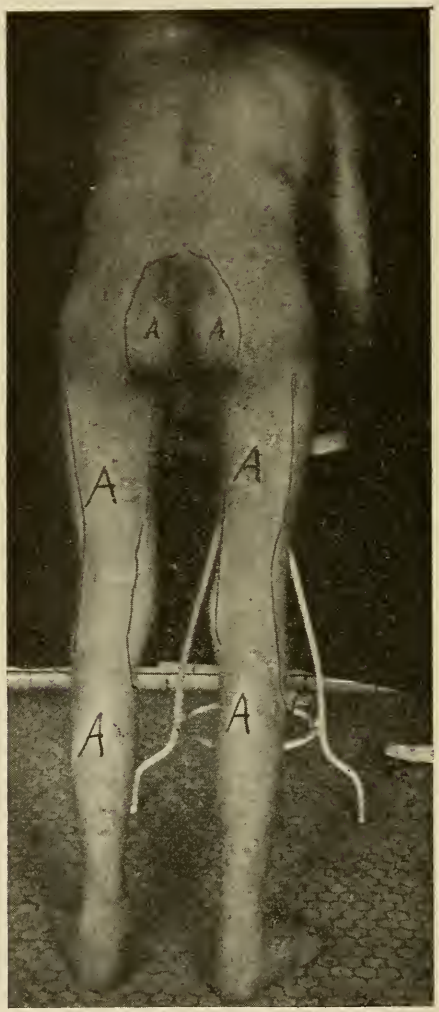


Fig. 131.—Sensory disturbance, weakness, and trophic disturbance following injury affecting the lower lumbar region of the cord. Posterior view. *A*, Area of anesthesia.

When disease or injury causes total interruption of the conduction of the cord, or if there is a complete transverse destruction of a segment or segments of the cord, there will be total loss of motion and sensation below the lesion, and a complete loss of

all reflexes—deep, superficial, and organic. Trophic disturbances, especially bed-sores, are apt to occur in the portions of the body below the lesion if there has been much involvement of the spinal cord.

Diagnosis.—It is important to diagnose the degree of injury to the spinal cord and nerve-roots, and to determine the exact

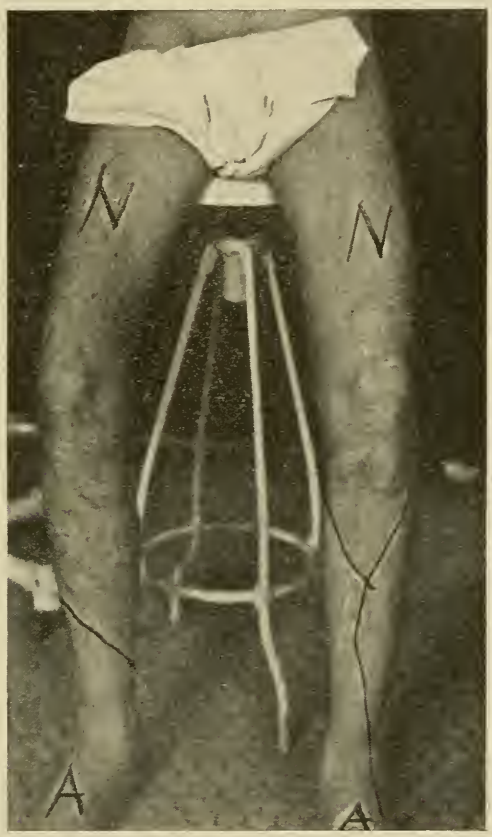


Fig. 132.—Sensory disturbance, weakness, and trophic disturbance following injury affecting the lower lumbar region of the cord. Anterior view. *N*, Area of normal sensation; *A*, area of anesthesia.

site of the lesion that affects the spinal cord. It is sometimes very difficult to determine, within the first few hours, the extent of injury that has been inflicted on the spinal cord. In some cases the symptoms of cord involvement do not occur for several hours or days after the injury; in other cases there is complete

loss of motion immediately after the injury, and sometimes marked interference with sensation, which rapidly disappears. In these two groups of cases there is usually no deformity; the symptoms are the result either of a hemorrhage into the spinal canal or of contusion and shock.

In the greater number of cases, immediately after the injury there is a disturbance or loss of motion and sensation and of bowel and bladder control. In extensive injury there are paralysis, loss of bowel and bladder control, and either complete or partial loss of sensation. In the less severe degrees of injury, motion, sensation, and bowel and bladder control are involved to a lesser extent. *x*-Ray examination and the distribution of the symptoms are useful as guides in locating the lesion. Usually the spinal cord is injured at the site of the fracture or dislocation, and is compressed either above or below the fractured or dislocated vertebræ.

Prognosis.—In cases of severe injury the prognosis as to life is very grave, the patients usually dying either soon after the injury or as the result of secondary infection from bed-sores or from the bladder. The less extensive and milder the symptoms, the better is the prognosis.

Treatment.—After a dislocation or fracture of the spine the patient should be moved as little as possible, all unnecessary manipulation of the injured parts being avoided. All active movements are to be prevented. Examinations are to be made with great care, and the injured parts should be supported in such a way as to preclude movement.

Operative interference is of no value in cases in which the symptoms point to complete transverse destruction of the cord, and is to be advised against. In those patients in whom the symptoms point to incomplete interruption of conduction in the cord there is a great diversity of opinion as to the advisability of performing early or late operation, or of avoiding operative interference entirely. No surgical procedure is justifiable during a condition of shock or if there are marked respiratory symptoms, greatly increased pulse-rate, or temperature disturbances. In those cases in which examination reveals the existence of pressure upon the spinal cord as the result of bony displacement,

etc., operation should be done as soon as the patient's general condition permits. When the symptoms point to a destructive condition of the cord itself, operation is not to be advised.

Most surgeons advise postponing operative procedures until after the second week, and then direct that only those patients be operated on in whom there is evidence of pressure on the cord.

In those cases that are operated on, and in those in which an operation has previously been performed, it is advisable to keep the back perfectly quiet by means of braces, a plaster-of-Paris jacket, etc., thus assisting nature in effecting union of the broken bones and preventing further deformity, with subsequent compression or traction of the cord. The greatest possible care should be taken to prevent the occurrence of bed-sores; infection of bladder is also to be guarded against, and the bowels should be emptied by means of enemata. Pain should be controlled by analgesics, and cramps and contractures are sometimes relieved by bromids and warm applications. The general health of the patient is to be looked after, and the muscle nutrition should be maintained by means of massage, etc.

TUMORS OF THE SPINAL CORD

Tumors occur very infrequently within the spinal canal. When they are present, they arise from the meninges about twice as often as they do within the cord itself. Tumors developing outside of the dura are very rare. Among the tumors that rise from the meninges are sarcoma, endothelioma, psammoma, syphiloma, tubercle, fibroma, myxoma, etc. Arising from the cord there are glioma and its mixed forms; outside of the dura, with the exception of those that arise from the vertebræ, are the lipoma and cysts. Malignant tumors are much more frequent than benign tumors in this region. Intramedullary tumors occur more often in the cervical and lumbar enlargement of the cord, whereas meningeal tumors occur chiefly in the dorsal region (Fig. 133).

Etiology.—Tumors of the spinal cord occur at any age: tubercular tumors appear more frequently in childhood, but the

other varieties occur at any age. In many cases a history of injury precedes the development of the tumor.

Pathology.—Tumors may be multiple, and often involve both the membranes and the cord. *Sarcomata* may appear either as solitary or multiple tumors; they develop from the nervous substance itself or from the membranes, and may be confined to

Fig. 133.—Spinal tumor, gliosarcoma, which was situated in lower dorsal and lumbar region of the cord; occurring in a case in the neurologic service at University of Maryland Hospital; successfully removed by Dr. R. Winslow.

the membranes, forming nodules, or they may spread diffusely over the surface of the cord. Sarcomata more commonly compress than destroy the cord, and usually extend rather rapidly; they are often associated with sarcoma of some other portion of the body.

Tubercle is more likely to develop within the cord, when it frequently occurs as a solitary tumor, having its origin usually

in the gray matter. There is a great tendency for these tumors to recur after removal, and there is also marked danger of infection of the meninges. Their presence is frequently complicated by a tuberculous meningitis. *Lipomata* are congenital. *Fibromata* originate within the meninges, and do not invade the cord; they are usually encapsulated and grow very slowly. *Gumma* commonly originates in the meninges, and infiltrates and frequently destroys the cord; it is diffuse and grows rapidly.

Myxoma never infiltrates the spinal cord; it is single, encapsulated, and of slow growth.

Glioma is an infiltrating tumor of the cord. It may or may not cause enlargement of the cord, and is distinguishable only microscopically. It usually grows rapidly and is frequently associated with sarcoma.

Tumors of the cord are usually small, but if they grow in a vertical direction, they may attain considerable size. Tumors of the meninges cause compression of the adjacent roots, and press upon the cord—some in front and some behind. The histologic changes show that the central parts of the spinal cord often suffer from the compression more than the external portion. The changes that take place within the cord are degeneration and softening in the region of the tumor.

Symptoms.—Root symptoms are usually the first evidence of the existence of a spinal tumor. These symptoms are characterized by intense, persistent, sharp, shooting pain in the distribution of one or two nerves. These pains are generally unilateral, but may be bilateral. The pain is often increased on exertion or on making violent unexpected movements. The location of the pain is dependent on the position of the tumor, it being felt in that part of the body related to the segment or nerve-roots first affected. In tumors developing within the cord, or even in some tumors developing from the meninges, these pains may be absent. The most persistent pains may later on disappear entirely, or the painful stage may persist for several months or even years before other symptoms pointing to direct involvement of the spinal cord manifest themselves. There is generally an area of hyperesthesia in the region of the trunk supplied by the segments, immediately above the affected one.

There is rarely any rigidity of the spinal column, and pressure does not increase the pain. If the growth increases in size and the compression destroys the nerve-roots or the spinal cord, two sets of symptoms arise: the one set in the distribution of the spinal segment or segments affected, and the other set due to interference through the affected segments of the impulses from above or below. The first set of symptoms consists of a flaccid paralysis of the muscles supplied by the nerves which originate in these segments, loss of the reflexes which take place through these segments, abolition or decrease of sensation in the portion of the body whose sensory nerves correspond to the affected segments, trophic disturbances, such as herpes, ulceration, vesicles, etc.

The symptoms due to interference with the transmission of impulses through the cord; if the site of the tumor is above the lumbar region, there will occur, in the portion of the body corresponding to the portion of the cord below the tumor, a spastic condition of the muscles, with weakness or paralysis, marked increase of the deep reflexes, and, if the tumor involves one side of the cord more than the other, the weakness, increased reflexes, etc., are more marked on that side.

Sensibility may remain unaffected for a considerable time, but if the compression continues, decrease and loss of sensation in the portion of the body below the lesion gradually develop.

In those cases in which the tumor affects principally one side of the cord, sensory disturbances, in which there is loss of temperature sense, pain sense, and partial tactile sense, will occur on the side opposite to that affected by the motor weakness, and on the side where the motor weakness exists there will be a loss of muscle sense, thus giving rise to the Brown-Séquard syndrome (Fig. 134).

There is usually an early impairment of the control of the bladder and rectum, and as the compression advances this is eventually completely lost. As soon as the sensory disturbances become marked there is a tendency to the development of bedsores.

In tumors that compress the lumbar cord the root symptoms

play a prominent part, for the nerve-roots are thickly grouped here and a small tumor may press upon a large number of roots.

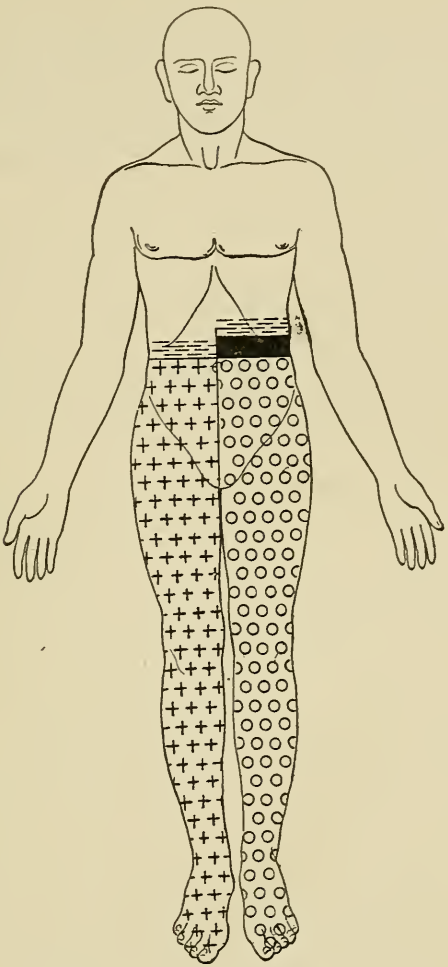


Fig. 134.—Brown-Séquard syndrome lesion involving spinal cord in middorsal region, left side. (▬) Area in which there is loss of all forms of sensation, vasomotor and trophic disturbance, loss of deep reflexes, and flaccid motor paralysis. (+) Area over which there is loss of tactile, pain, and temperature sense. (o) Area over which there is loss of muscle sense, partial tactile sense, and in which there is spastic motor paralysis. (– – –) Area of cutaneous hyperesthesia.

The pain affects the lower abdomen, the pelvis, and the legs. The symptoms caused by compression of these roots, as well as of

the lumbar enlargement, are a flaccid, atrophic paralysis, with loss of reflexes, decrease or loss of sensation, bowel and bladder disorders, and trophic disturbances.

Tumors of the cauda equina cause great pain in the region of the sacrum, radiating into the perineum, bladder, and the sciatic nerves. To these pains are added atrophic paralysis, limited to the bladder and the muscles supplied by the nerves of the sacral plexus; disturbance of sensation of the perineum and the segments of the body corresponding to the sacral segments of the cord.

In multiple and diffuse tumors the symptoms usually point to involvement of several segments of the cord.

The symptoms of tumor of the cord resemble those of a transverse myelitis of gradual onset, combined with persistent neuralgic pains in the distribution of the posterior roots of the affected segments.

The course of the disease is a chronic one. It generally begins with pain, which is persistent, and which is usually unilateral, weakness on one side of the body below the lesion, followed by a paraplegia, to which are later added the symptoms of a progressive transverse myelitis. Death occurs from infection, cystitis, cachexia, etc., the disease running its course in from a few months to several years, depending upon the character of the growth and the rapidity of its development.

Diagnosis.—The diagnosis of tumors of the spinal cord should be made as early as possible, for it is only before destructive conditions of the spinal cord have developed that we can hope to secure a favorable outcome after the growths have been successfully removed. The diagnosis of the presence of a growth must first be made; second, the probable character of the growth must be determined; third, the location of the growth must be ascertained, and fourth, it must be learned whether it is single or multiple. Tumor of the cord must be differentiated from spinal syphilis: in spinal syphilis the onset is more rapid and the course is intermittent, with a tendency to sudden exacerbations and remissions. The symptoms usually indicate lesions at various sites in the spinal cord or brain.

The physical examination, the Wassermann test of the blood

and spinal fluid, and the result of antisyphilitic medication all assist in making the differential diagnosis.

The differentiation between tumors of the spinal cord and caries of the spinal column is made by the presence of rigidity, tenderness on pressure, increase of pain on motion, general constitutional disturbances, etc., which are present in spinal caries and absent in tumor of the cord. *x*-Ray examination is also of value.

The presence of the tumor or the history of a previous operation for removal of tumor of some portion of the body is always of importance.

The variety of tumor can rarely be determined until after its removal. By taking into consideration the fact that some growths are of more rapid development than others; that some occur more frequently within the cord substance, whereas others originate more commonly within the meninges, it is possible, before operating and during life, to hazard a tentative opinion as to the character of the growth.

The location of the tumor is to be diagnosticated by the distribution of the symptoms corresponding to the affected segment, and by the presence of symptoms pointing toward interference with impulses through the cord at the affected segment. Whether one or more tumors exist will be shown by the presence of symptoms pointing to segmental involvement at more than one level of the cord.

It is important to determine whether we are dealing with a vertebral, meningeal, or medullary tumor. In vertebral tumors *x*-ray examination, deformity of the spinal column, pain on movement of the spinal column, and the fact that these tumors are usually secondary to malignant tumors elsewhere usually guide one to a correct diagnosis. Meningeal tumors generally begin with root pains, flaccid paralysis in the distribution of the affected segment, and gradual development of symptoms pointing toward compression of the cord; furthermore, meningeal growths rarely extend upward, so that the upper limit of the segmental symptoms remains unchanged. In tumors of the cord substance itself the root symptoms are not prominent and may be absent entirely, the symptoms resemble those of a

chronic myelitis, the course is usually irregular, and there may be a sudden appearance of new symptoms, due to hemorrhage into the tumor.

In diagnosing the location of a spinal tumor it must always be remembered that the pain and anesthesia, which are the most reliable indications of the level of the tumor, will be the same in a lesion of a segment as in a lesion of its nerve-roots at a point somewhat lower down within the spinal canal. It is, therefore, advisable to examine the cord at a slightly higher level than the lesion would lead one to expect to find it. It is also important to learn opposite which spinous processes the various segments of the cord are situated.

Chipault has given a practical set of rules for determining the relation of the segments of the cord to the spinous processes of the vertebræ. In the cervical region, add 1 to the number of the vertebræ: this will give the segment opposite to it; in the upper dorsal region add 2; from the sixth to the eleventh dorsal vertebræ, add 3. Opposite the eleventh dorsal process and the space below it are the lower three lumbar segments; opposite the twelfth dorsal spinous process and the space below it are the sacral segments. The spinal cord ends opposite the body of the first lumbar vertebra: below this the spinal canal is occupied by the cauda equina. This set of rules applies only to adults. In infancy and childhood the spinal cord extends lower down into the spinal canal, and these figures are not, therefore, altogether accurate.

Prognosis.—In tumors of the cord the prognosis is, as a rule, grave, unless surgical intervention be undertaken. If they are of tubercular nature, the outcome in practically all cases is fatal. In a few benign tumors there may be a spontaneous arrest of growth. Cord tumors of syphilitic origin may respond to treatment. The greater number require surgical intervention. Diffuse intramedullary tumors cannot be removed, and the outcome is fatal. If the diagnosis is made early and surgical intervention is attempted the prognosis is more favorable.

Treatment.—In those cases in which pain is a prominent symptom it will be found that while the coal-tar products at first afford some relief, morphin will eventually have to be used.

In cases in which syphilis is suspected, antisyphilitic measures may be tried, and if prompt relief does not follow, recourse should be had to operative intervention.

When the tumors are multiple, or when the growth is secondary to a tumor in some other portion of the body, and likewise in those cases that originate in the vertebral body, operation is contra-indicated. When but a single growth is present, and if this can be localized with some degree of accuracy, early operation is to be advised. In the hands of a skilful surgeon the operation is not a very difficult one, and can be undertaken with the assurance that very little damage is done even if no growth is found. In those cases in which there is a meningeal growth that has not infiltrated but only compresses the cord, the outcome is good if the operation is undertaken early and is properly done.

SPINAL MENINGITIS

Acute spinal leptomeningitis is seldom a primary, isolated disease. It may be of traumatic origin. As a rule, the infection extends upward and involves the membranes of the brain. The inflammation may be limited to the spinal meninges, as after injury, various diseases of the spinal column, especially tuberculosis and malignant diseases. It may also occur as an extension of an otitic meningitis, after surgical operations upon the spinal column or the cord, as a complication in spinal anesthesia, as the result of extension of a syphilitic process, from bed-sores, exposure to cold, etc. Most commonly it is secondary to the presence of an infectious agent in the blood, following pneumonia, typhoid fever, tonsillitis, septicemia, etc.

Pathology.—Upon opening the spinal canal an exudate of serous, fibrosanguineous, or purulent character is seen deposited in the network of the pia-arachnoid.

The cerbrospinal fluid is turbid. The meninges are covered with the exudate, and in places are more or less adherent. The blood-vessels are distended, and many small hemorrhages may be found. On microscopic examination there will be seen congestion of the finer blood-vessels of the meninges and the arteries entering the cord, a serous exudation about the blood-vessels,

both in the pia and in the cord, and swelling, congestion, and thickening of the pia. The nerve-roots and the superficial tissues of the spinal cord are also usually involved in this inflammatory process, and show some evidence of degeneration, which is most marked about the blood-vessels. The posterior portions of the spinal membranes are generally more severely affected than the anterior.

In tubercular meningitis the spinal fluid is usually clear and there is little or no exudate; if any is present it is of a gelatinous nature. Miliary tubercles may be found in the membrane, especially in the neighborhood of the blood-vessels. The pathologic changes are usually diffuse, but if the disease extends downward from the brain, the inflammatory changes may be limited to the upper portion of the spinal canal.

Symptoms.—The onset is usually sudden, with chill, high fever of irregular type, and marked constitutional disturbances. Accompaning or following these symptoms there is acute pain in the back, which is increased by movement. The pain radiates about the body and into the extremities, and there is stiffness in the back and neck, which may result in opisthotonus. Tonic contractures of the muscles of the back, abdomen, and extremities are present. Pain and stiffness are increased when attempts are made to move the patient. In addition, there are hyperesthesia of the skin; exaggeration of the cutaneous and abdominal reflexes; clonic tremors of the trunk and extremities, especially upon handling the patient; the Kernig sign, etc. As the disease advances weakness or paralysis develops, disturbance of sensation takes place, bowel and bladder control is interfered with, and there is a tendency to the developement of bed-sores. Vasomotor disturbances are common, and there may be cardiac and respiratory symptoms.

The **course** is acute and usually rapid; within a week there is marked involvement of the respiratory and cardiac functions. In the majority of cases the meningitis extends into the cranial cavity, and cerebral symptoms develop. In other cases the progress may be slower, and several weeks may pass before the symptoms become alarming. Some patients pass through the acute stage, which may last several weeks, without manifesting

alarming symptoms; then the meningitis becomes chronic, the symptoms continuing to be present for several months.

Diagnosis.—The diagnosis is made by lumbar puncture and examination of the spinal fluid, in conjunction with symptoms pointing toward involvement of the spinal meninges.

Prognosis.—The prognosis of acute meningitis is always grave. In the tubercular variety recovery does not seem possible. Those forms that are secondary to pneumonia, typhoid fever, etc., if they remain limited to the spinal meninges, usually recover. The form due to the Diplococcus intracellularis will generally recover under proper treatment.

Treatment.—Absolute rest in bed. It is advisable for the patient to lie upon the side, rather than upon the back. An ice-cap or counterirritation should be applied to the spine. General constitutional disturbances should receive proper treatment. The bowels should be kept well open, and elimination encouraged by the free drinking of water. Pains are to be relieved and restlessness is to be combated by such drugs as the bromids, codein, opium, etc.

The patient is to be guarded against the development of bedsores. Spinal puncture is to be employed both for diagnostic and for therapeutic purposes. In those cases due to the Diplococcus intracellularis antimeningococcus serum is to be given.

CHRONIC MENINGITIS

Rarely acute meningitis may become chronic. In these cases the meninges become opaque, thickened, and generally adherent to each other and to the cord. The changes may be localized or extend throughout the entire cord. In the majority of cases a low-grade chronic myelitis, limited to the periphery of the cord, is also present.

Another form of chronic meningitis is of syphilitic origin. In this variety there is a gummy or gelatinous exudate in the meshes of the pia and arachnoid, very often involving the nerve-roots as well. Thickening of the vessels and infiltration of round cells are usually present. The lesions, both of the meninges and of the cord, may be scattered irregularly throughout the entire cord, and may vary in extent.

Symptoms.—The onset of the disease is slow, and it usually runs a prolonged course. There is an absence of general constitutional symptoms. Pain is present, radiating in the distribution of the spinal nerves, and hyperesthesia, stiffness in the back, a tendency to convulsive movements, disturbance of sensation, weakness or paralysis, disturbance in the control of the bowels and bladder, etc., are also seen.

Diagnosis.—The diagnosis is made from the history of preexisting acute attacks, or if the condition is of a syphilitic nature, by examination of the blood and spinal fluid.

Treatment.—The cautery applied to the spine, massage, long-continued warm baths, the alternate application of hot and of cold water to the spine, the promotion of the general nutrition of the patient by proper food, fresh air, and tonics are the indications. Inunctions of mercury and potassium iodid internally are of value in all forms of the disease. In those cases that are of syphilitic nature the intravenous administration of salvarsan, and if the spinal fluid gives a positive Wassermann reaction the intraspinous injection of salvarsanized and mercurialized serum are indicated.

HYPERTROPHIC PACHYMENINGITIS

This is a chronic inflammation, affecting chiefly the inner layer of the dura mater, producing a laminated deposit of fibrous tissue on the dura, and causing it to become markedly thickened.

Etiology.—Exposure to cold, overexertion, trauma, alcoholism, tuberculosis, and especially syphilis must be included among the causes. In the majority of cases several factors are responsible. A history of syphilis, alcoholism, and trauma is present in the greater number of cases.

Pathology.—There is a fibrous thickening of the dura, the newly formed membranes being arranged in layers. The pia and arachnoid play a very important part in the inflammation, which frequently originates in these membranes. As a result of the thickening of the meninges there is produced compression of the cord and of the nerve-roots. Not uncommonly hemorrhages occur into this newly formed tissue or upon its inner sur-

face. New blood-vessels are formed in this tissue, and these are very thin and rupture easily. This tissue causes compression of the spinal cord and its blood-vessels, and as a result myelitis or softening takes place. The exudation may be confined to the upper or lower part of the cervical enlargement, or it may extend upward to the pons or downward, or may be limited to the lumbar region.

Symptoms.—These are caused by the meningitis, by the compression of the posterior roots, and by compression of the cord. The distribution of the symptoms is dependent upon the extent and location of the exudate. There is usually pain in the back, between the shoulders, in the neck, and even in the head. Stiffness and tenderness are often present in the cervical region or in other parts of the back. Severe neuralgic pains following the course of the nerves that originate in the affected region are the earliest and most prominent symptoms. As the disease advances, which it does very slowly, there follow paralysis of the atrophic type in the distribution of the nerves, disturbances of sensation in the corresponding area, and, if the disease progresses further, to these local symptoms are added the evidences of spinal compression, such as spastic paralysis of the legs, disturbances of sensation, bowel and bladder disorders, etc.

The disease is further characterized by irregular exacerbations of symptoms.

If the upper cervical region is involved, the first symptoms to appear are local pain and stiffness, with neuralgic pains in the distribution of the cervical nerves. If the lower cervical region is involved, in addition to the local symptoms pain will occur in the distribution of the ulnar and median nerves. If the exudate appears first in the lumbar region, in addition to the local symptoms there will be neuralgic pains in the lower extremities.

Diagnosis.—If the disease affects the cervical region the diagnosis is not difficult. It must be differentiated from diseases of the vertebral bodies and from tumors. From disease of the vertebral bodies it is differentiated by *x*-ray examination, by the usual absence of tenderness, by the chronic course, and by the intense neuralgic pains, followed by atrophic paralysis long before the occurrence of symptoms pointing to interference

with conduction through the cord. From tumors of the cord it is differentiated by the presence of muscular rigidity, intense pain, and the paralytic symptoms in the distribution of the affected nerves before the appearance of evidence of interference with conduction in the spinal cord.

Course.—The course of this disease is very slow; it may last for many years and cease to progress at any time.

The **prognosis** is fairly good if the diagnosis is made early.

Treatment.—The bowels should be kept freely open, and elimination induced by abundant drinking of water. The general health should be maintained by nutritious food and hygienic surroundings. Pain may be relieved by pyramidon, codein, etc. Mercurial inunctions and large doses of potassium iodid are indicated. Some patients seem to do well upon the salicylates. Daily warm baths are also advisable. Counterirritation to the spine over the suspected area by means of the actual cautery is useful. Tincture of iodin or cantharides locally seems to promote the absorption of the exudate. Surgical intervention is not usually possible, as there is generally a close adherence between the membrane and the cord, and its removal would result in injury to the cord.

MYELITIS

Myelitis is a diffuse or disseminated process of inflammation and softening in the spinal cord.

Etiology.—This disease is not associated with any particular age, although occurring most frequently in middle life. In most cases infection is the cause of the disease. The inflammation of the cord may be the only evidence of infection, or a myelitis may develop secondary to some infectious disease, such as influenza, measles, typhoid fever, tonsillitis, erysipelas, or pneumonia, or it may also follow syphilis, tuberculosis, pregnancy, meningitis, injury or operation on the cord, or the result of the direct extension of inflammatory conditions in the neighborhood of the spinal cord. Exposure to cold, overexertion, and injuries, such as falls and blows upon the back, all predispose to this condition. Chronic intoxications may also be a predis-

posing factor or may be followed by myelitis. Males are more frequently affected than females.

Pathology.—The inflammatory lesion may be limited to one or two segments, or the lesion may be disseminated, with more or less normal tissue intervening. The lesion may be diffuse, and show a tendency to extend from one segment to another, either upward or downward or in both directions. In other cases the infection is irregular in distribution and in its advancement. Occasionally the infection may remain localized to a single area. Macroscopically, the gross appearance of the cord may not be altered, although some of the vessels may appear injected and the cord opposite the lesions may appear softer than normal. On section the spinal cord is seen to be of semifluid consistence, all differentiation between the gray and the white matter being obliterated. It presents either a yellowish or a reddish appearance. In less severe cases differentiation between gray and white matter may still be possible, and the section may present points of congestion and irregular areas of yellowish or reddish color. Microscopically, there are seen congestion of the vessels and infiltration of the tissue by leukocytes and small cells. The cells of the cord and the nerve-fibers appear to be in all stages of degeneration. There is a proliferation of neuroglia cells, and granular bodies are found throughout the degenerated tissue. In addition to these changes in the cord it is frequently possible to isolate various pathogenic organisms, such as streptococcus, staphylococcus, pneumococcus, etc. In some cases, as a result of the disintegration and inflammation, there may be total destruction of the spinal cord—this occurs in rapidly fatal cases. In the milder cases the destruction is less complete, and if the inflammation subsides, scar tissue replaces the nervous elements that have been destroyed. Those cells that have not been seriously damaged may resume their function, the cells that have been seriously injured or destroyed are incapable of regeneration. As a consequence those functions of the body that depend for their activity upon the portions of the cord which have been destroyed are permanently lost. In some cases of myelitis the presence of pathogenic organisms cannot be demonstrated; this may be due to the fact

that the organisms that have set up the inflammatory process have subsequently disappeared, or that the inflammatory process is secondary to a toxic condition or to an injury. In many cases inflammation of the membranes of the cord complicates the picture of the disease.

Symptoms.—The disease usually comes on more or less acutely; less frequently there is a subacute onset, days, weeks, or months elapsing before all the symptoms are evident. In some cases there is a slight rise of temperature as well as a general feeling of malaise, a more or less rapid pulse, etc.; within a few hours, days, or weeks symptoms appear that point to direct involvement of the cord itself. The character, severity, and distribution of the symptoms depend upon the location and extent of area involved. In some cases there is a complete transverse myelitis, in others an incomplete transverse myelitis, and in still others a disseminated myelitis. The symptoms arising from these lesions are of two kinds: First, those due to interference with the functions of the portion of the cord that is affected, and, second, those due to interference with the transmission of impulses upward and downward through the affected area.

The first group of symptoms consist of paralysis with atrophy, reaction of degeneration, loss of the reflexes, decreased sensation or anesthesia, and vasomotor, trophic, and secretory disturbances in the parts of the body over which the affected segments of the cord preside.

The location and severity of these symptoms depend upon the segment of the cord invaded and the extent of the involvement.

The second set of symptoms are due to interference with the transmission of stimuli through the affected area of the cord. They occur in parts of the body below the site of the lesion, and consist of weakness or paralysis; exaggeration of the tendon-reflexes; increased muscle tone, giving rise to a spastic condition; contractures and spontaneous spasmodic movements. Babinski's sign is frequently present. The control of the bladder and rectum is usually lost. Sensibility may be decreased or lost—usually all forms of sensation—tactile, temperature, pain, and muscle sense—are equally involved.

Trophic Disturbances.—When pressure is continued over an area in which sensation is lost a destructive condition is produced that gives rise to bed-sores. The latter may be superficial in nature, but sometimes they penetrate very deeply. They are especially prone to occur over the sacrum, trochanters, heels, etc. Vasomotor disturbances may also occur, and there may be edema, coldness and blueness of the skin, an increase or decrease of the secretion of sweat, etc.

If a transverse myelitis exists in the cervical region, the first set of symptoms occurs in the upper extremities and the second set involves the trunk and lower extremities. If a transverse myelitis exists in the dorsal region of the cord, the upper extremities will escape, the first set of symptoms occurring in the distribution of the nerves of the affected segment, and the second set affecting the trunk below the lesion and the lower extremities. If a transverse myelitis occurs in the lumbar and lumbosacral region, the entire picture is changed, owing to the fact that practically all the symptoms are of the first type. In those cases in which the transverse myelitis is not complete the condition manifests itself in incompleteness of the individual symptoms. In those cases of myelitis in which numerous inflammatory areas are present at various sites in the spinal cord the symptoms depend upon the location of these foci and upon their extent. In cases in which there has been a complete transverse destruction of the spinal cord the disease picture differs in so far that there will exist a complete flaccid paralysis with loss of all reflexes below the site of the lesion.

Diagnosis.—Myelitis is to be differentiated from multiple sclerosis, tumor of the spinal cord, compression of the spinal cord, and vascular disease of the spinal cord.

From *multiple sclerosis* the condition is readily differentiated by the presence, in the former, of nystagmus, speech disturbance, intention tremor, etc., and the absense of these symptoms in myelitis.

From *tumor of the spinal cord* myelitis is differentiated by the much slower onset and the absence of fever, constitutional symptoms, and of a history of some preceding infection. From *compression of the spinal cord* it is differentiated by the history,

x-ray examination, and mode of onset. From *vascular diseases of the spinal cord* it is differentiated by the presence or previous history of general vascular disease; by the sudden onset of the condition, without fever, pain, or constitutional disturbances; by the fact that the disease rapidly attains its maximum and then either remains stationary or improves, or by the occurrence of subsequent attacks.

Course.—Myelitis usually develops within a period of from a few hours to several weeks or months. The disease generally runs a slow course. If the patient lives, a certain amount of improvement may be expected.

Prognosis.—In mild cases the prognosis is fairly good; in the severer cases, however, the prognosis as to total recovery is absolutely unfavorable, and as to life is very grave, owing to the fact that there is great tendency for infection to occur through the bladder, rectum, or through bed-sores. Those cases that are secondary to infectious diseases and occur in young, healthy individuals, and those in which there is but little anesthesia seem to show the greatest tendency to recover.

The more acute and at the same time incomplete the development of the symptoms, the more favorable, in general, is the prognosis. The early appearance of rapidly spreading bed-sores and of complete paralysis of the bladder and rectum is usually an indication of an unfavorable outcome.

Treatment.—First and most important is absolute rest. The patient should be confined to bed and moved as little as possible. The greatest care should be exercised to prevent the occurrence of bed-sores, and the skin should be carefully looked after. Proper attention should be given to the bowel and bladder functions. If the condition follows an infectious disease, elimination by the intestines, skin, and kidneys should be encouraged. Counterirritation along the spine is useful, care being exercised not to blister any of the anesthetic areas. The patient should be turned carefully from side to side, and pressure kept off all bony prominences by properly adjusted pillows or rings. The patient should not be allowed to sit up to empty the bowel or bladder. Internally, laxatives, salol, the salicylates, or aspirin may be administered. Urotropin may also be given,

but the effect of this drug on the kidneys must be watched. For the spasmodic conditions and for the cramps in the muscles warm cloths may be applied, together with bromids internally. In some cases quinin seems to do good. Care should always be exercised to avoid injuring the patient in the anesthetic areas. After the disease has reached its height and the patient begins to improve, warm baths and massage seem to help. If the patient is confined to bed, every effort should be made to prevent the formation of bed-sores. The bowels and bladder should be carefully attended to. In those areas in which there is an atrophic flaccid paralysis, galvanic and faradic stimulation hasten recovery. Later, when the patient begins to walk, overfatigue and injury should be guarded against, and in suitable cases braces, etc., should be prescribed.

HEMATOMYELIA

This condition is due to rupture of a vessel with extravasation of blood into the cord substance.

Etiology.—Injuries, such as follow severe blows, falls, dislocation or fracture of the vertebræ, or direct trauma to the cord as the result of penetrating wounds, etc., are by far the commonest causes of hemorrhage into the cord. Forced flexion of the vertebral column forward is especially apt to induce a hemorrhage into the cord. Violent muscular exertion, such as lifting heavy weights, may be followed by rupture of a vessel. In individuals of the hemorrhagic diathesis spontaneous hemorrhage may occur, and it may also take place after typhoid fever, in the puerperium, after severe labor, etc. Hemorrhage into the cord may likewise occur in areas which are already diseased, as in the presence of tumors, syringomyelia, myelitis, etc.

Pathology.—Spinal hemorrhage almost always occurs in the gray matter. When, however, it is due to direct injury, such as fracture, dislocation, or penetrating wounds, the hemorrhage may involve any portion of the cord and may also take place into the spinal canal. The gray matter, being supplied by larger vessels, is especially predisposed to hemorrhage.

Any segment may be affected, but the enlargements, particu-

larly the cervical, are more frequently involved. The hemorrhage may extend through the whole central gray matter, or be limited to one side or even to one horn. It is seldom so extensive as to give rise to swelling of the cord, and it is especially likely to spread in the longitudinal direction of the cord. Externally, the cord may show no evidence of the existence of a hemorrhage. Occasionally the cord may be ruptured and the hemorrhage reach the surface of the cord. In some cases inspection of the cord may reveal little or nothing, whereas in others marked disintegration of the cord substance and hemorrhage throughout the subdural space may be seen. On section, if the patient dies within a short time, the clot will be found surrounded by broken-down cord tissue, etc. If the patient survives for some time, the clot may be partially absorbed, and the tissue about it be stained a yellowish red, the nervous elements appearing to be in all stages of disintegration. In those patients who survive a secondary degeneration develops in the cord above and below the injured segment.

Symptoms.—The symptoms depend upon the location of the hemorrhage and, to a certain extent, upon the cause. If the hemorrhage follows a dislocation or fracture of the vertebral bodies, in addition to the symptoms pointing to involvement of the cord there occurs evidence of local injury. As a rule, there is deformity and muscular rigidity, with fixation of the back. Movement causes pain at the point of injury and in the distribution of the nerves which are compressed or lacerated. *x*-Ray examination will usually reveal the extent of injury in so far as the bony structures are concerned. In some cases no deformity is present and the skiagram reveals no abnormality, but the clinical symptoms nevertheless point to extensive involvement of the spinal cord.

In those cases in which hemorrhage takes place into diseased tissues of the cord the symptoms of hemorrhage are added to those of the preëxisting disease. When the hemorrhage follows a penetrating wound, there frequently occurs a purulent inflammation of the meninges as well as of the cord itself. In these cases, in addition to the symptoms resulting from involvement of the cord, there are marked pain and rigidity of the

back, due to the meningitis. The symptoms pointing to involvement of the cord vary according to the site of the hemorrhage. There is usually a sudden interruption of conduction in the spinal cord. If the patient is standing, he falls. Paraplegia, loss of control of the bowel and bladder, and sensory disturbances develop below the site of the lesion, and vasomotor and trophic disturbances soon appear.

The symptoms increase in severity and extent during the first few hours. The distribution of the symptoms, of course, depends upon the location of the hemorrhage within the cord: if the lesion is in the cervical enlargement, there will be a flaccid paralysis of the upper extremities, with vasomotor, trophic, and secretory disturbances; anesthesia in the areas corresponding to the segments that are affected; disturbances or loss of the tendon-reflexes, and disturbance of electric reaction of the affected muscles. Spastic paralysis affects the part of the body below the lesion, and there are increase of deep reflexes, decrease of the superficial reflex, bowel and bladder disturbances, complete or partial anesthesia, the development of bed-sores, and vasomotor and trophic disturbances. If the lumbar enlargement is affected, there is flaccid paralysis of the legs, with absence of the tendon reflexes, atrophy of the muscles, and complete or partial anesthesia, corresponding to the segments that are affected, bowel and bladder disturbances, etc. If the lesion is limited to the three lower sacral segments, the symptoms consist of loss of control of the bowel and bladder functions, with complete relaxation of the sphincters, loss of sexual power, and anesthesia limited to the perineum, posterior part of the scrotum, or posterior half of the labia, and extending in a heart-shaped area over the sacrum. In those cases in which the hemorrhage is irregular, or in which one side of the cord is principally affected, the symptoms are not symmetric—the paralysis and sensory disturbances vary in distribution and degree on the two sides of the body. In cases in which the lesion is limited to one-half of the cord, Brown-Séquard's syndrome occurs; this is characterized by the following combination of symptoms: On the side of the lesion there is spastic paralysis, with increased tendon-reflexes and decrease or loss of muscle sense in the parts below

the lesion; a narrow zone of anesthesia about the body, at the level of the lesion, in which all forms of sensation are affected. On the side opposite the lesion there is no paralysis; slight increase of tendon reflexes; loss of the sense of touch, temperature, and pain, up to a line about the body somewhat lower than the area of anesthesia.

In most cases there is pain in the back; this, however, is not severe or persistent unless the spinal meninges are involved. There may also be slight rise in temperature within the first few days or weeks.

Course and Prognosis.—The onset is usually sudden. The symptoms may become more intense for several hours, and then remain stationary, or the patient's general condition may grow much worse. A temperature of from 100° to 101° F. usually occurs. Some cases develop extensive bed-sores and gradually become weak, emaciated, and eventually die. In others after a few days the symptoms begin to subside. If death does not rapidly ensue in those cases in which the hemorrhage is not extensive and is not due to fracture, dislocation, penetrating wounds, or secondary to some diseased tissue of the cord, as a rule improvement takes place within a few days or weeks. The symptoms that are the result of compression gradually disappear. Improvement goes on only to a certain point, for all the symptoms due to destruction of the substance of the cord are permanent. The later danger in these cases is from toxemia, infection, etc., as the result of bed-sores, cystitis, etc. The prognosis in those cases secondary to injury of the spinal column and penetrating wounds, etc., depends largely upon the degree and severity of the symptoms: the higher the lesion, the more unfavorable is the prognosis both as to life and as to degree of recovery. Those symptoms that are due to pressure, if not too long continued, usually disappear, whereas those that are due to destruction of the cord tissue are permanent.

Diagnosis.—This condition is differentiated from *myelitis* by the fact that in myelitis the onset is never so sudden—there are usually premonitory symptoms. From *hemorrhage into the spinal meninges* the disease is differentiated by the fact that in the former there occur symptoms of irritation, such as severe

pain in the back, tenderness of the muscles to pressure, stiffness of the back, and symptoms of compression of the roots and cord which develop slowly. Spinal puncture is also an aid to diagnosis. From *myelomalacia* the condition is differentiated by the freedom from temperature on the second or third day, by the history of the absense of exciting causes of hemorrhage, by the presence of a low blood-pressure, and the evidence of general vascular disease. From *acute poliomyelitis* it is differentiated by the fact that this disease is usually ushered in by a feverish stage, by the absence of sensory disturbance, and by the peculiar distribution of the motor paralysis.

Treatment.—Absolute rest for several weeks. All movements should, as far as possible, be prevented. Treat the cause. The development of bed-sores should be guarded against. The bowels and bladder should receive proper attention. The diet should be light and non-stimulating. In those cases in which the occurrence of a meningeal or epidural hemorrhage is suspected, lumbar puncture or operative interference is advisable.

MYELOMALACIA

This condition is due to occlusion of the arteries by thrombi, with subsequent softening and disintegration of the portion of the spinal cord supplied by the affected vessels.

Etiology.—Among the causes may be mentioned: Cardiac disease; atheroma of the larger vessels; syphilis, old age, states of general weakness, malnutrition, chronic diarrhea, infectious diseases, anemia, the puerperal state, overexertion, strains, etc.

Pathology.—When accompanied by the transudation of blood-cells the area of softening may be red; when the condition has been one of simple death of tissue it may be white; when the process is not observed until after a fatty degeneration has ensued the area will be yellow. Microscopic examination shows cells and fibers in various states of disintegration; neuroglia cells and fibers in a state of hyperplasia; granular débris; and in cases of red softening, the presence of many red blood-cells.

In the course of time the products of disintegration are absorbed, being replaced by connective tissue, and leaving, in

place of the nervous tissue, sclerotic areas that are irregular in outline and variable in extent. These areas are frequently wedge shaped, with the base directed toward the periphery of the cord.

Blood-vessels usually show changes in their wall. Their lumen is diminished, either as the result of a true endarteritis or from infiltration of the adventitial coat or thickening of the intima.

Symptoms.—Symptoms are dependent altogether upon the location of the lesion. They are preceded for some time by symptoms pointing to general vascular disturbance. Disturbances due to involvement of the cord come on more or less acutely within a few hours. There is no rise of temperature, and usually no constitutional disturbances occur.

The symptoms are of two kinds: the first are due to the destruction of the elements of the spinal cord at the point of the lesion, and consist of atrophic paralysis, with reaction of degeneration in the muscles supplied by the cells situated in the anterior horns, which have been destroyed; loss of the reflexes, which takes place through the affected segments; paresthesia and anesthesia in the area of skin connected with the affected segment. The second set of symptoms are produced by interference with the transmission of impulses from above and below through the affected area. They consist of spastic paralysis or weakness, with increased reflexes below the lesion; disturbance of control of the bowel and bladder; disturbance or loss of sensation; trophic disorders, such as the development of bed-sores, etc.; a tendency to muscular contraction and spasms, etc. In those cases in which the lesion is situated in the lumbar enlargement of the cord the symptoms are all practically of the first type. The level of the sensory disturbances is the best indication as to the location of the lesion.

Diagnosis.—The disease is to be differentiated from hemorrhage into the cord and from myelitis. From *hemorrhage into the cord* it is distinguished by the absence of the usual causes of the hemorrhage and the absence of slight rise in temperature; the presence of low blood-pressure; the slower onset, etc. From *myelitis* it is not always possible to differentiate this condition.

Myelomalacia shows a more acute onset, an absence of temperature, and is more apt to occur after a debilitating or infectious disease than during the course of such a disease.

Course.—The onset is usually more or less sudden, the disease attaining its height in a few hours, and remaining at this point for several days, after which gradual improvement takes place up to a certain point, the patient then entering a chronic state similar to that of chronic myelitis.

The **prognosis** depends upon the patient's general condition, upon the extent of the lesion, and upon the subsequent care the patient receives. The prognosis as to life is usually fairly good, as to improvement fair, but as to total recovery very unfavorable.

Treatment.—The patient should be kept in bed, great care being taken to avoid the development of bed-sores. The bowels and bladder should be carefully looked after. The general nutrition of the patient should be improved by proper food, fresh air, massage, etc. Internally, potassium iodid in small doses and strychnin in proper doses several times a day should be given.

SENILE PARAPLEGIA

In old age a condition may occur that is characterized by a progressive weakness in the lower extremities, with some stiffness, to which may be added disturbance of sensation and weakness of bowel and bladder control. This condition is especially apt to occur in those who are feeble and who have a more or less general degeneration of the cardiovascular system.

The patients usually complain of a sensation of numbness and tire easily. There is difficulty in retaining the urine. The disease progresses slowly. The patients experience difficulty in ascending steps, there is a tendency to drag the feet, and an uncertainty in the gait. There may be a general tremulous state. Occasionally the patients will also complain of weakness and numbness of the upper extremities.

Pathology.—This condition is probably due to disturbances of circulation of the cord and to sclerotic changes in the neighborhood of the vessels, especially in the white matter.

Prognosis.—As to improvement, the outlook is fairly good, as most of these patients respond well to treatment.

Treatment.—Attention is to be given to the patient's general condition. Nourishing food, general massage, cardiac stimulants, and properly administered hot and cold douches to the trunk and spine are all useful in the treatment of this condition.

LANDRY'S PARALYSIS (ACUTE ASCENDING PARALYSIS)

This disease is characterized by an acute ascending paralysis, beginning in the lower limbs, and rapidly ascending to involve the body, arms, and respiratory muscles. It is attended by fever and constitutional disturbances, death generally ensuing within a few days.

Etiology.—The disease is believed to be caused by some acute toxemia or infection. Up to the present time there is no consensus of opinion as to the source or character of the causative agent.

Pathology.—No definite pathologic changes are present. In some cases a disseminated inflammatory condition of the central nervous system has been described; in other cases an inflammatory condition in the anterolateral columns has been seen; anterior horn inflammation and peripheral nerve inflammation have also been described. The most constant changes seem to be inflammatory and vascular in nature and to involve the gray matter of the spinal cord.

Symptoms.—The disease is characterized by a general feeling of malaise, slight fever, which is accompanied by weakness, and then paralysis, begininng in the lower extremities. This paralysis is flaccid in type, and extends rapidly to involve the muscles of the trunk, next of the arms, then of the neck, and last involving the muscles of deglutition, of speech, and of the face. When the arms are affected, it begins in the fingers and gradually advances upward. The bladder and rectum usually remain normal. There is a loss of the tendon reflexes. As the disease progresses marked respiratory disturbances appear and death usually supervenes with symptoms of asphyxia. This occurs in some cases within three days after the onset, and in others within a week or two. Paresthesias are usually present.

There may be other disturbances of sensation, although these are not prominent as a rule. There does not seem to be any decrease in the size of the affected muscles, although this may be due to the fact that death so rapidly follows the onset of the disease that atrophic changes have not had time to manifest themselves. The electric reaction may or may not be disturbed.

The **prognosis** is very grave, as it is extremely rare for a case to recover.

CONGENITAL SPINAL MALFORMATION (SPINA BIFIDA; RACHISCHISIS)

A congenital fissure in the spinal column is due to an arrest of development which produces merely a defective closure of the spinal column and spinal membranes, or the simultaneous formation of a tumor, due to a hernial protrusion of the meninges through the fissure in the form of a subcutaneous swelling (meningocele). The process usually involves the cord, which it also displaces outward (meningomyelocele). The absence of the posterior wall of the spinal column may be complete or partial, only the latter possessing clinical interest. This incomplete closure is usually situated in the lumbosacral region, less frequently in the cervical region, and very rarely in the dorsal region. There is no posterior closure of the dural sac and the meninges at the fissure, so that the bottom of the sac is occupied by a ventral portion of the pia and arachnoid; above this is a red, velvety membrane, composed of a vascular plexus and nerve elements; this represents the more or less distorted lower segments of the spinal cord (Fig. 135).

Myelomeningocele is the commonest form of spina bifida. In this deformity the spinal cord reaches its posterior wall, whereas the accumulation of fluid lies ventral to it. In most cases of spina bifida the development of the lower segments of the spinal cord is more or less impaired. The cord is flattened out when it enters the cyst formed by the meninges, or becomes attached to the walls. These tumors are, as a rule, situated in the lumbosacral region of the spinal column. They vary in size, their diameter ranging from one or two inches to eight to ten inches. They are elastic and fluctuating. The defect of the vertebræ

may be felt or demonstrated by radiography. Spina bifida is frequently accompanied by other malformations.

The **symptoms** arising from the pathologic changes in the lumbosacral cord and nerve-roots are, to a great extent, congenital, but may not become evident until later in life. There usually occur paralysis of the leg muscles, atrophy, reaction of degeneration, paralysis of the bowels and bladder, disturbance

Fig. 135.—Spina bifida (case of Dr. A. Shipley).

or loss of the knee-jerks, trophic disorders, such as perforating ulcer, disturbances of sensation, etc.

Prognosis.—The prognosis is very grave. These patients usually die very early in life, death being due to ulceration of the skin over the sac, to secondary infection, or to rupture of the cyst with the escape of cerebrospinal fluid. The more marked the paralysis, the greater is the danger to life.

Treatment is purely surgical. The outlook for recovery is very unsatisfactory.

DISEASES OF THE BRAIN

Pachymeningitis

PACHYMENINGITIS EXTERNA

Pachymeningitis externa, or inflammation of the external surface of the dura mater, does not occur as an independent affection.

Etiology.—It develops in conjunction with affections of the bones of the skull. These may be of traumatic origin, or secondary to caries, osteomyelitis, syphilis, erysipelas, tumors of the cranial bones, inflammation spreading intradurally, etc.

The **symptoms** depend upon the cause. The symptom that points toward involvement of the dura is pain, and if the dura is much depressed, there is evidence of intracranial pressure.

The **prognosis** depends upon the cause, and the **treatment** is surgical.

PACHYMENINGITIS INTERNA

This is an inflammation of the internal surface of the dura.

Etiology.—The condition affects especially the aged and chronic alcoholics. It occurs also in chronic inflammatory brain affections accompanied by atrophy of the brain, in chronic insanity, tuberculosis; arteriosclerosis, and in those suffering from chronic heart and kidney diseases. It may follow sunstroke and the acute infectious diseases, and may complicate syphilis, pernicious anemia, leukemia, hemophilia, etc. Trauma to the skull is the most frequent exciting cause.

Pathology.—A thin, vascular, inflammatory, membranous exudate appears on the inner surface of the dura, more commonly over the parietal lobes, it may be very extensive. If the condition persists for some time the membrane presents a laminated appearance, as if successive layers were formed at different times. This membrane is very vascular, the vessels being extremely delicate and rupturing easily. In most cases

hemorrhage takes place into this membrane, with the formation of hematomas; these are usually thin, but with the successive formation of layers pressure is produced against the brain, which gives rise to symptoms.

In the course of time this tissue becomes organized and may undergo calcareous degeneration. Two theories as to the cause of the formation of this exudate have been advanced: the first is that the exudate is inflammatory in origin, and, the vessels being very delicate and thin, rupture easily, giving rise to hemorrhage; the second holds that first a hemorrhage occurs, which later becomes organized and forms this membranous exudate, and that subsequent hemorrhages are responsible for subsequent layers. When the hemorrhage causes an increase of symptoms the condition is known as pachymeningitis interna hæmorrhagica.

Symptoms.—The disease may exist for years without giving rise to any marked symptoms. In many cases the symptoms of the brain affection mask the manifestations of the pachymeningitis, and the true condition is only discovered at autopsy. The symptoms that can be attributed directly to the pachymeningitis are restlessness, dull headache, difficulty in concentrating the thoughts, mental slowness, hemiplegic or epileptic seizures, comatose states that may last for long periods, evidence of intracranial pressure, slow pulse, respiratory disturbances, and choked disc. There may be local tenderness of the skull to percussion, etc.

The **course** of the disease is usually a very slow one, and may be greatly prolonged or may rapidly prove fatal.

The condition is usually characterized by a gradual remission of symptoms, with a period during which the patient is almost completely relieved of the symptoms pointing to involvement of the dura. Then occurs a more or less sudden return of the symptoms. These attacks are particularly apt to follow trauma to the head, sudden exertions, acute illness, etc.

Prognosis.—The prognosis is dependent upon the cause. In some cases death follows rapidly after an acute exacerbation of symptoms, but in most cases the course is prolonged, with remissions and exacerbations of symptoms.

Diagnosis.—The diagnosis is rarely made during life. In individuals suffering from chronic mental affections, chronic alcoholism, etc., the existence of a pachymeningitis hæmorrhagica may be suspected when there are present restlessness, dull headache, slowness of the mental processes, transient comatose states, or epileptiform seizures.

In doubtful cases, especially after trauma to the head, lumbar or cranial puncture may enable one to make a diagnosis.

Treatment.—Where the condition is suspected, the patient should be put to bed and guarded against injury. Ice-bags should be applied to the head. Elimination by the bowels and kidneys is to be brought about. The diet should be light, and in plethoric individuals bleeding is indicated. Lumbar puncture or, where the evidence is sufficient, opening of the skull and removal of the membrane and clot constitutes the surgical treatment.

MENINGEAL HEMORRHAGE

Hemorrhage may occur either between the dura and the skull or between the dura and the brain, or it may take place beneath the pia. *Extradural hemorrhage* usually comes from the meningeal arteries, and is generally the result of injury to the skull. *Intradural hemorrhage* may arise from laceration of meningeal arteries, of the veins in the pia or of veins opening into the sinuses. Either of these forms of hemorrhage may be caused by trauma without coincident rupture of the continuity of the bones. *Subarachnoid hemorrhages* occur usually after severe injuries in which the brain substance is also involved: this is the type of hemorrhage that usually occurs in infants after prolonged or difficult labor (Fig. 136), and is sometimes seen in individuals who have sustained a blow on the head the force of which was widely diffused. The site of the hemorrhage usually corresponds to the site of the injury, but arterial laceration through contrecoup takes place on the opposite side or on both sides.

The amount of blood effused may reach 200 grams or more. The blood may form one large clot, or may be diffused as a thin layer over the surface of the brain; if it is subarachnoid, it may be scattered irregularly in thin layers beneath the arachnoid,

over the entire surface of the brain, or be limited to several lobes or convolutions.

Extradural Hemorrhage.—*Symptoms.*—The symptoms of *extradural hemorrhage* depend upon the location of the hemorrhage. As a rule, there is a history of injury, followed more or less quickly by the onset of a comatose condition, sometimes immediately and at other times after the expiration of several hours. Weakness or paralysis occurs upon the opposite side to the injury. The pupil on the paralyzed side is contracted, whereas that on the side of the injury is dilated.

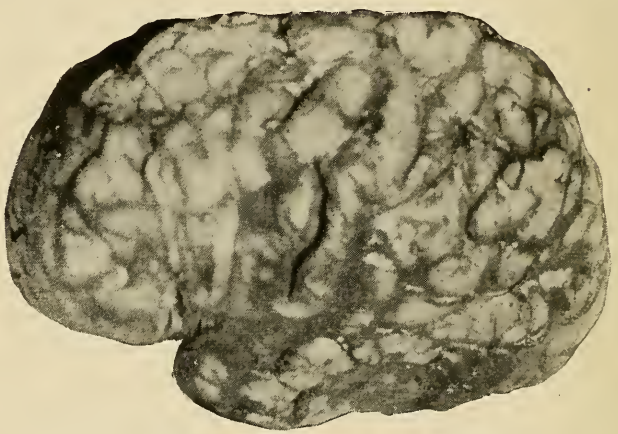


Fig. 136.—Subpial hemorrhage following labor.

If the patient regains consciousness, weakness or paralysis, disorders of speech, disturbance of sensation, etc., may be discovered.

Diagnosis.—The diagnosis is made from the history of injury and from the symptoms. Lumbar puncture in these cases does not show the presence of blood in the cerebrospinal fluid, but may disclose a brownish-yellow staining of the fluid.

Treatment.—These cases should be operated upon as soon as the diagnosis is made. The lacerated vessels should be ligated and the clot removed.

Symptoms of Intradural Hemorrhage.—In these cases, either with or without fracture of the skull, there may be a stuporous state due directly to the injury. If stupor occurs, the patient

may regain full consciousness, only to lose it again after a certain time. After several hours restlessness may develop, or there may be delirious excitement, which soon passes into a state of stupor that may deepen into coma. There is evidence of intracranial pressure, the pulse becoming slow, and the breathing irregular and stertorous. Choked disk sometimes develops. There are usually at first unilateral spasms, followed later on by paralytic symptoms. There is generally a rise in temperature, and the pupil on the affected side may be dilated. Localized tenderness of the skull may be present. In some cases the symptoms are more indistinct and vague, the patient complaining of a transient giddiness, pain in the head, loss of memory, and mental slowness.

The **symptoms of subarachnoid hemorrhage** are quite similar to those just enumerated. In this latter group of cases the symptoms gradually progress and increase for hours or even days, and then subside very slowly and only partially disappear. There is a gradual loss of consciousness, and headache, slow pulse, vomiting, respiratory disturbances, and rise of temperature occur. To these symptoms are added others pointing toward interference of function of the portion or portions of the brain that are affected, such as paralysis, loss of sensation, aphasia, hemianopsia, etc.

Course.—Following an injury, strain, or compression the patient may become unconscious. He may remain in this state or may regain consciousness, or within a few hours or days after the onset of the affection there may be a gradual loss of consciousness or a stuporous state may develop. In some cases a progression of symptoms develops, pointing to involvement of successive areas of the brain. These are at first irritative and later paralytic. In some cases the symptoms are accompanied by a delirious state, slow or rapid pulse, disturbance of the respiratory rate, rise of temperature, etc. The coma may deepen, the pulse become rapid and irregular, and death ensue. In other cases the patient may regain consciousness and gradual improvement in the symptoms appear for a time, and then the condition may remain stationary.

Prognosis.—The prognosis depends upon the extent and

location of the hemorrhage. As to life it is usually good, except in cases of very extensive hemorrhage. The prognosis as to recovery of function depends upon the amount of brain tissue that has been destroyed.

Treatment—In those cases in which the symptoms point to a localized collection of blood, trephining the skull over the affected area, with ligation of the lacerated vessels, is to be done as soon as possible. When the symptoms point to diffuse extravasation of blood, the patient should be put to bed, the head slightly elevated, and ice-caps applied to the head. Internally, calomel, followed by a saline purgative, is to be administered. The bladder should be emptied at regular intervals. The diet should be very light. If there is evidence of increased intracranial pressure, a decompression operation is indicated.

MENINGITIS

This is an inflammatory condition of the pia-arachnoid, usually affecting also the brain, especially the cortex, so that in most cases a meningo-encephalitis is present. The onset is generally acute, and there is, as a rule, a diffuse distribution, although in some varieties the inflammation begins in a limited area and extends gradually.

Etiology.—Inflammation of the meninges is usually due to the presence of a microörganism. A purulent meningitis may be due to streptococci or to staphylococci, pneumococci, etc. A special form of meningitis, known as epidemic cerebrospinal meningitis, or spotted fever, is caused by the Diplococcus intacellularis of Weichselbaum.

Microörganisms may reach the meninges from a neighboring area of suppuration, or they may come by way of the blood, or lymph-stream as a part of a general infection, or following injuries to the skull with open wounds. The inflammation may be due to injuries and infections of the vertebral canal caused by the entrance of infecting organisms, with subsequent extension to the cerebral membranes; following erysipelas or purulent otitis with caries of the bone; extension of the inflammation from the nasal cavities, orbit, or pharynx; following

acute infectious diseases or eruptive fevers, such as typhoid, influenza, etc. Other types may occur secondary to tuberculosis, syphilis, chronic toxemia, etc. Basing the classification on the etiology, at least five varieties of meningeal inflammation may be said to occur:

1. Septicemic or secondary meningitis.
2. Epidemic cerebrospinal meningitis.
3. Tubercular meningitis.
4. Syphilitic meningitis.
5. Serous or toxic meningitis (wet brain).

SEPTIC OR SECONDARY MENINGITIS

This form of meningitis is always secondary to some primary cause; as a consequence, before the manifestations of meningitis appear symptoms pointing to the causative agent will have occurred.

Etiology.—Among the commonest forerunners of this form of meningitis are diseases of the middle ear, the nose and its accessory sinuses, injury to the skull, operations upon the head, erysipelas of the face, abscesses about the head and face, septic endocarditis, and osteomyelitis. In addition to these causes must be mentioned the acute infectious diseases, septicemia, and prolonged gastro-intestinal or pulmonary diseases in very young children. Following any of these conditions pathogenic organisms may gain access to the cerebral membranes and set up an inflammatory condition.

Pathology.—There is first a hyperemia of the pia mater, which later becomes cloudy in appearance. The cerebrospinal fluid is at first turbid, but later becomes purulent. Pus is found at first over the sulci of the brain, and later the entire surface is more or less covered. The superficial tissues of the cortex undergo inflammatory changes and small hemorrhages occur. The ventricles become somewhat dilated and contain a sero-purulent exudate. In some cases, especially those secondary to middle-ear or nasal diseases, inflammation may be limited at first to a portion of the brain, finally spreading. As a general rule the membranes of the spinal cord become involved.

Symptoms.—If a meningitis develops during the course of any of the aforenamed conditions, there is usually a more or less sudden exacerbation of the general constitutional symptoms which are present as the result of the causative affection; thus chills may occur, and there is usually a rise in temperature; the pulse becomes more rapid, and restlessness develops; to these symptoms are added evidences of involvement of the meninges and brain. Severe headache occurs, giving rise to great restlessness, and in young children there is more or less constant rolling of the head from side to side. In some cases this last is so constant as to cause the rubbing off of the hair from the back of the head. The patient becomes very irritable, and sleep is usually disturbed, and in some cases almost impossible. Infants and young children frequently awake from a light sleep with a scream (the cephalic cry); later, if the disease progresses, delirium alternates with stupor, finally merging into coma. There is a general state of hyperesthesia, evidenced by avoidance of bright light and loud noises, and cutaneous sensitiveness appears. Muscular twitchings, cervical rigidity, opisthotonos, and convulsions sooner or later make their appearance. Added to these symptoms are the manifestation of involvement of the cranial nerves, optic neuritis, nystagmus, unequal pupils, loss of the pupillary reflex, grinding of the teeth, facial paralysis, etc. The tendon-reflexes are usually increased, and there is disturbance of control of the rectal and vesical sphincters.

The clinical picture of the case depends largely upon the cause and upon the portion of the meninges first affected. In those cases secondary to injuries to the skull the portions of the meninges that are first affected are those closely related to the site of injury, and the symptoms that occur are those due first to irritation of that portion of the brain, and later, as the affection extends, to the irritation of other portions. Those cases that are secondary to nasal sinus disease and to middle-ear disease usually begin with symptoms pointing toward involvement of the adjacent cerebral tissues. In the septic cases, in which the organism generally gains access by the blood-stream, the convexity of the brain is generally first involved, and as the inflammation becomes more or less diffuse there occur severe

headache, marked delirium, great restlessness, hypersensitiveness, and often convulsions. There is marked disturbance of the mental state, manifesting itself in a loss of interest in the surroundings, difficulty in comprehension, and intellectual slowness. These are followed by symptoms pointing to involvement of the base of the brain, such as cervical rigidity, cranial nerve involvement, etc. This is particularly true of those cases which occur either during the course of or follow the acute infectious diseases, such as influenza, typhoid fever, the eruptive diseases, etc.

When meningitis occurs secondarily in very young children, who seem to be especially susceptible, before the close of the cranial sutures, hydrocephalus develops if the disease lasts more than ten days. In addition to the general constitutional disturbances and symptoms pointing to involvement of the meninges, there is usually a rapid emaciation.

The blood count shows leukocytosis, from 12,000 to 30,000 leukocytes being present. Examination of the urine frequently reveals the presence of albumin, sometimes of sugar, and rarely of casts. Spinal puncture yields a fluid that is turbid, containing cells, leukocytes, and microörganisms; it generally flows under increased pressure.

Course.—In some cases all the symptoms appear with great rapidity, the disease running its full course in a few days. In other cases the symptoms are of slower onset, the irritative disturbances being the first to appear, to be followed later by the paralytic symptoms: in these cases the disease may run a slower course, lasting several weeks.

The **prognosis** in this condition is very serious: few recoveries take place, and symptoms pointing to brain destruction, such as mental weakness, paralysis, blindness, etc., frequently remain as sequelæ.

Diagnosis.—The diagnosis is made from the history of the preceding illness, together with the symptoms of meningeal inflammation. Spinal puncture should be made, and if examination of the fluid reveals the presence of pathogenic organisms or of abnormal cellular constituents the diagnosis is confirmed. Meningitis must be differentiated from the acute infectious

diseases, especially pneumonia and typhoid fever, particularly in the case of children, in whom these diseases may begin with headache, delirium or stupor, vomiting, general hyperesthesia, convulsions, etc.

A careful physical examination, spinal puncture, and a regular pulse-rate, despite the increase in temperature, will usually clear up the diagnosis.

Acute otitis media, especially in children, may be accompanied by cerebral symptoms that suggest a meningitis. A careful examination of the cerebrospinal fluid will reveal the presence or absence of pathogenic organisms, abnormal cells, or both.

Treatment.—This consists, first, of the proper treatment of the underlying cause, so that the occurrence of meningitis may be avoided. In those cases in which meningitis already exists, the focus of infection should receive attention. The patient should be kept in a well-ventilated, darkened, quiet room and ice-caps be applied to the head. The bowels should be kept well open. The pain is to be controlled by codein and morphin in proper doses, and the restlessness and convulsions diminished by the adminstration of bromids. Temperature should be kept down as far as possible by sponging with cool water. In those cases that require stimulation, caffein, alcohol, and strychnin are to be used. The withdrawal of cerebrospinal fluid by lumbar puncture at times seems to afford the patient some relief. Urotropin in proper doses has been recommended, but seems to be of little value.

EPIDEMIC CEREBROSPINAL MENINGITIS

This is an infectious, but not a contagious, disease. It is due to the Diplococcus intracellularis of Weichselbaum. The disease usually occurs in epidemics during the winter and spring, but sporadic cases may occur at any time. Children and young persons are most commonly affected. Injuries may act as the exciting cause, but the organism is generally introduced by way of the lymphatics from the nose and pharynx. Persons living in crowded districts and amid unhygienic surroundings are more likely to be attacked than those who live amid better conditions.

Pathology.—Early in the disease hyperemia and congestion of the pia mater occur, followed by a serofibrinous exudate which later becomes purulent. These pathologic changes affect the convexity and base of the brain, the surface of the cerebellum, and the spinal meninges. The cerebral ventricles contain a turbid and purulent fluid. The cortex of the brain also becomes affected, the vessels becoming dilated. The superficial tissues are congested, and slight hemorrhages and small collections of pus are found throughout the superficial layers. The cerebral nerves are embedded in and infiltrated by the exudate. The inflammatory process may extend along the auditory nerves to the internal ear, and set up a purulent inflammation.

Symptoms.—The symptoms usually appear suddenly. Chill may or may not occur, and there is a rise of temperature, which varies from 101° to 105° F. The patient complains of a feeling of illness, restlessness, headache, and backache; the pulse is rapid and full, and within a few hours or days vomiting may occur. The headache becomes severe, and vertigo occurs. The patient appears to be very ill, is restless, becomes sensitive to light and sound, unable to sleep, and complains of general cutaneous sensitiveness. Convulsions may occur, especially in early childhood. Within a short time the neck becomes stiff, and if the disease advances, the head is retracted and a state of opisthotonos develops. Spasms of the muscles of the extremities and face may occur, and the pupils may be dilated, unequal, and not react to light. Grinding of the teeth, strabismus, facial paralysis, etc., may also be present. Fever persists, the temperature often rising to 104° or 105° F., and the patient becomes actively delirious. The abdomen is retracted, the bowels are constipated, and there is retention, with later incontinence, of urine. The delirium alternates with stupor and coma, and is worse at night and when the temperature rises. About the third day, in the majority of cases, an eruption appears. This is diffused more or less over the entire body, and may be erythematous, urticarial, petechial, or purpuric. Herpes occurs early, the vesicles forming chiefly upon the lips, the face, the ears, and the extremities. The vesicles last only a few days

and then dry up. Irritation of the skin leaves dark-red spots—the tache cérébrale of Trousseau. Contractures develop early, and later on paralysis occurs. In those cases that do not respond to treatment there is rapid emaciation. The cranial nerves are almost always affected, and optic neuritis, oculomotor paralysis, deafness, facial paralysis, difficulty in swallowing and in articulation are prone to occur. The fever runs a very irregular course, and great variations in temperature are possible. The pulse is usually rapid and irregular, and may vary at times from 40 to 120 or even 140 beats a minute. The respiration may also be irregular in rate and in type. At first the patient is restless and actively delirious, but later in the disease he either becomes comatose or assumes a state of indifference.

There is usually a marked leukocytosis, the white-cell count ranging from 10,000 to 30,000. The urine generally contains albumin and casts. Examination of the cerebrospinal fluid reveals the presence of an intracellular diplococcus. The cerebrospinal fluid may be clear at the first puncture, and be turbid upon subsequent punctures.

Course.—The course of this disease varies in different epidemics and in different individuals. Some cases run a very rapid course. In these there are active delirium, convulsions, great prostration, paralysis, coma, and death within a few days. In other cases in which the symptoms are not so severe there may be a remission in five or six days, during which the patient seems very much better, but which is followed shortly by a recrudescence of all the symptoms, which may be more severe and result in a fatal termination within a few weeks.

In those cases in which a correct diagnosis has been made and the proper treatment instituted there is usually a marked improvement after one or two injections of Flexner's antimeningococcic serum. In some cases, even with this treatment, there is a persistence of mental inertness, general hyperesthesia, cervical rigidity, headache, etc. In such cases there is usually an obstruction in the communications between the ventricles and the general subarachnoid space.

Prognosis.—Since the discovery of Flexner's serum the prognosis is fairly good, provided the diagnosis is made early. The

prognosis is also governed by the severity of the case. In spite of proper treatment some cases succumb rapidly to the disease. In others recovery is incomplete, certain symptoms, such as deafness, paralysis, blindness, headache, etc., becoming more or less permanent.

Diagnosis.—The condition is to be differentiated from other forms of meningitis, typhoid fever, uremia, etc. The presence of herpes, the history of the case, and the fact that spinal puncture yields a fluid containing an intracellular diplococcus will readily enable one to make a correst diagnosis.

Treatment.—The patient should be kept in a cool, quiet, darkened room, and ice-bags should be applied to the head and spine. The bowels must be kept freely open. If the temperature rises above 102.5° F., the body should be sponged with cool or cold water every two or three hours. In those cases in which there are convulsions, restlessness, etc., the bromids or opium in proper doses frequently repeated are of use. The patient's strength is to be supported by light, nourishing food. Water should be given freely.

As soon as the diagnosis has been confirmed by withdrawing from 15 to 30 c.c. of cerebrospinal fluid, from 15 to 30 c.c. of Flexner's antimeningococcic serum is to be injected intraspinally, the larger doses being given in the more severe cases. The cerebrospinal fluid should be examined daily. The diplococci disappear as the case improves, and a leukocyte count should be made daily, as there is a gradual decrease in the leukocytosis if the case responds to treatment.

Injections should be given daily until the diplococcus disappears from the fluid. If there is a recurrence of the symptoms, injections must again be made. In some cases in which there is a persistence of the cerebral symptoms, such as coma, headache, etc., it may be necessary to make a ventricular puncture, and if the fluid thus withdrawn contains diplococci, to inject the antimeningococcic serum directly into the ventricle. After the acute symptoms have subsided the patient should receive a nourishing diet, tonics, and an abundance of fresh air.

TUBERCULAR MENINGITIS

This disease is due to the invasion of the cerebral membranes by tubercle bacilli: it is always secondary to tuberculosis in some other portion of the body, most frequently in the lungs, lymphatic glands, bones, etc.

Etiology.—This disease occurs more frequently in early life, particularly between the second and the fifteenth year. It is seen only rarely in infancy and after the fortieth year. Among the predisposing causes are trauma, the acute infectious diseases,—such as measles,—whooping-cough, alcoholism, catarrhal disease of the intestines, overexertion, etc.

Pathology.—The route by which the tubercle bacilli reach the cerebral membranes is not certainly known: they may be carried to the brain by the blood-stream and through the lymphatic system. Miliary tubercles are deposited on and in the cerebral membranes, there is a serofibrinous and gelatinous exudate, and rarely pus is present. The exudate and the tubercles are deposited in larger or smaller masses at the base of the brain, about the anterior and posterior perforated spaces, and from here they extend into the fissures and up over the surface of the brain, in some cases going backward to the under surface of the medulla, pons, and cerebellum, and in others extending to the spinal membranes. When the process involves the convexity of the brain, it is more extensive in the fissures and around the blood-vessels. Miliary tubercles occur not only in the exudate, but also in parts that are free from inflammation. They present a gray white, shiny, nodular appearance, and vary in size from almost a pin-point to large masses the result of coalescence of smaller areas. The ventricles are usually distended and filled with a serum in which fibrin and blood are found.

The choroidal plexuses are also generally studded with tubercles, and a degree of hydrocephalus, depending upon the amount of interference with the circulation from the ventricles to the general subarachnoid space, and upon the extent of ossification of the cranial sutures, is present. The exudation also surrounds the nerves, giving rise to a neuritis. As a rule, the cerebral

substance itself is affected, resulting in diffuse or localized encephalitis, which is frequently of a hemorrhagic nature. Areas of softening in the substance of the brain and the basal ganglion are frequently present, and may be traced to obliteration of small vessels as the result of a tubercular arteritis.

Symptoms.—Before the symptoms pointing to the true nature of the disease occur there is usually a period, lasting for some days or even longer, during which manifestations pointing to general disturbance appear.

Prodromal Symptoms.—In a child that has previously been healthy, but pale or poorly nourished, or in adults who have had or have been exposed to tuberculosis, there occurs a period during which the individual is irritable, manifests no interest in his surroundings, is indifferent, and perhaps at times confused. The sleep is more or less disturbed and interrupted by disagreeable dreams, and there are more or less persistent headache, diffuse pain, and sensitiveness, the patient reacting abnormally to mild forms of irritation. There may be loss of appetite, constipation, loss of weight, and sometimes vomiting. After a shorter or longer period the second stage, or stage of irritation, sets in.

Stage of Irritation.—At this time the headaches become more severe, delirium develops, and convulsions may occur. Restlessness and grinding of the teeth develop, the patient sleeps lightly and wakes with a scream, the head becomes retracted, there is great sensitiveness to light and sound, and the head is rolled from side to side. No control is exercised over the rectal and vesical sphincters, and there may be tonic contractures of some or of all the muscles of the body, muscular twitchings, etc. The cranial nerves are usually affected. The pupils are irregular, and strabismus, nystagmus, vertigo, facial paralysis, etc., occur. Throughout this stage, which may last from a few days to two weeks, there is constant evidence of the irritative condition of the meninges and cortex. The temperature in these cases varies from subnormal or slightly above normal to 105° or 106° F. The pulse and respiration are irregular, and do not usually correspond with the temperature. Emaciation is rapid, and in very young children a hydrocephalus commonly develops,

in which the increase in size and shape of the head is accompanied by a slight bulging of the eyeballs. During this stage the patient gradually grows weaker, and the active delirium is replaced by a state of stupor. The skin is dry, and tache cérébrale is well marked; usually choked disk is present. Early in this stage, in spite of the severity of the symptoms, there may be remissions, during which the patient seems to be better, but as the last stage approaches these intervals become less frequent and are of shorter duration.

Toward the end of this stage the stupor deepens into coma. the irritative symptoms subside, and are replaced by those of a paralytic nature.

The third, or paralytic, stage is characterized by the presence of coma and of paralysis of some of the cranial nerves and of the extremities. The opisthotonos is marked; the abdomen is retracted; the pupils are dilated and fail to react to light; fever is more or less constantly present; the pulse becomes more rapid and weak, and the respiration is irregular and often of the Cheyne-Stokes variety. Emaciation becomes more marked, and control over the sphincters is completely lost. The patient dies in this state from exhaustion, or a convulsive seizure may occur and terminate the illness.

Course.—The disease usually begins with evidences of a general illness, which is more or less marked in a child or an adult who either has tuberculosis or has had some other illness, or who is pale and whose resisting power is poor, or it may appear in a child who is previously apparently healthy. In other cases the disease is ushered in with a convulsion, and the first stage may last from a few days to several weeks.

The second stage, which is characterized by symptoms of irritation and hyperesthesia of the central nervous system, lasts from a few days to two weeks, rarely longer.

The third stage, which is marked by symptoms pointing to loss of function of the central nervous system, lasts from a few days to one or two weeks. The average duration, from the onset of the disease to its fatal termination, is about four weeks.

Prognosis.—The prognosis is very unfavorable: a few re-

coveries have been reported, but in the vast majority of cases the outlook is hopeless.

Diagnosis.—Differentiation must be made from other forms of meningitis, vascular disturbances of the brain, hysteric conditions, uremia, catatonic states occurring in various forms of mental disease, cerebral trauma, etc.

There is little difficulty in arriving at a correct diagnosis if the history of the condition is taken into consideration and a spinal puncture is made. The cerebrospinal fluid is usually increased in amount, may be clear or slightly turbid, and shows an increase in its cellular constituents, varying in number from 15 or 20 to 150 per cmm. These cells are generally of the small mononuclear type. There is an absence of all microörganisms except the tubercle bacillus, which can be found in most cases if diligently sought.

Treatment.—The patient should be placed in a quiet, darkened, and well-ventilated room, and be given as much nourishment as he can comfortably take. Pain and convulsions should be controlled by the use of sedatives. As very little can be done for this condition, it is important that its occurrence be prevented, as far as possible, by proper attention to the care and surroundings of children who display a tubercular tendency or who already have tuberculosis.

SYPHILITIC MENINGITIS

This form of meningitis occurs during the second stage of syphilis, and is due to the presence of the Spirochæta pallida. It usually appears within the first few years after infection has taken place. Injury to the head, mental exhaustion, worry, etc., may act as exciting causes of this condition.

Pathology.—In this form of meningitis a gummy deposit is usually seen in the subarachnoid tissues, and round-cells infiltrate the cerebral tissues. As a rule, the exudate is first deposited in the region of the two cerebral peduncles, and spreads irregularly, especially around the blood-vessels. The nerves at the base of the brain are also usually surrounded and even infiltrated by the exudate. This gummy exudate may be

localized and form a tumor-like mass, or may be very diffuse in its distribution. In some few cases it may develop first on the convexity of the brain.

Histologic examination of the exudate shows it to consist of granulation tissues rich in cells and containing many small vessels. This exudate is never purulent. The round-cells infiltrate the walls of the vessels and the epineurium of the nerves. As the result of this infiltration the walls of the vessels become thickened, and their lumina decreased or obliterated. The nerve becomes swollen as the result of the infiltration, and the individual fibers may become atrophied as the result of the pressure.

Symptoms.—A person affected with a syphilitic meningitis will complain of more or less persistent headache, which may be very severe, and which usually grows worse toward evening and at night. Attacks of vomiting, vertigo, disturbance of consciousness, or convulsions may also occur. Quite early in the condition it is noticed that there are disturbances of memory, and that the individual is more or less indifferent. There may be attacks of prolonged stupor or of excitement, during which the patient is confused and delirious. To these symptoms are usually added evidences of interference with the functions of the cranial nerves.

When the oculomotor nerves are affected, ptosis, paralysis of any or all of the external ocular muscles, and pupillary disturbances appear. As this disease may involve the whole or only a part of the oculomotor nerves, there may be either a limited or an extensive involvement of the ocular muscles. The optic nerve is also frequently involved, giving rise to either unilateral or bilateral optic neuritis or choked disk. The first nerve may be involved, causing loss of the sense of smell. If the seventh nerves are involved, facial paralysis and interference with hearing occur. It is not unusual for the cranial nerves to be irregularly affected. The involvement of the cranial nerves varies considerably, and is inconstant. To these symptoms are frequently added those of involvement of the cerebral vascular system. These usually develop late in the disease. As a rule, there is little or no increase in temper-

ature. The clinical picture varies greatly, owing to the fact that the exudate may be limited or very extensive in its distribution.

Course.—As a rule, the onset of the disease is very insidious, headache preceding the other symptoms for a considerable period. In a few cases the onset may be very acute. The disease usually pursues a more or less chronic course, characterized by repeated remissions and exacerbations, periods occurring during which the patient seems almost well.

Prognosis.—The prognosis is generally favorable if the condition is recognized early. It is only after the disease has existed for some time and the nerves have undergone atrophic changes that recovery cannot be hoped for. In those cases in which repeated apoplectic attacks have occurred the outlook is unfavorable.

Diagnosis.—This condition is differentiated from *tumor of the brain* by the history, the variability of the symptoms, the distribution, and the irregular involvement of the structures at the base of the brain. Examination of the blood and spinal fluid and the effect of treatment are additional aids to diagnosis.

It is very difficult at times to differentiate this disease from *general paresis*. Usually the diagnosis of general paresis can be made from the history of very slow onset, the primary infection having occurred many years previously from the prominence of the mental symptoms and the absence of headache. In some cases differentiation between the two diseases can be made only as the result of treatment, syphilitic meningitis usually responding well to treatment, whereas general paresis responds only indifferently if at all.

From other forms of meningitis this disease is differentiated by the absence of temperature and the result of examination of the cerebrospinal fluid.

Treatment.—Energetic antisyphilitic treatment should be instituted at once whenever there is even a suspicion of a syphilitic brain condition. The treatment should consist of the intravenous injection of salvarsan. Three days after this mercurial inunctions should be given daily for three weeks, and

potassium iodid in appropriate doses internally. At the end of four weeks a second injection of salvarsan should be given, and mercurial inunctions and potassium iodid again resumed. In those cases in which the cerebrospinal fluid yields a positive Wassermann reaction, intradural and in some cases intraventricular injections of salvarsanized, mercurialized serum should be given. The antisyphilitic treatment should be continued until the blood and spinal fluid yield a negative Wassermann reaction. The general health of the patient should receive attention. The diet should be properly supervised, and diversion, fresh air, and exercise prescribed.

In those cases in which the symptoms point to a localized pathogenic state which does not yield to antisyphilitic treatment, surgical intervention is justifiable. It is most important that syphilitic infection should be promptly and thoroughly treated so as to prevent the occurrence of subsequent syphilitic diseases of the nervous system.

SEROUS MENINGITIS—MENINGISMUS

This is a condition characterized by many of the symptoms of meningitis, the cerebrospinal fluid being usually increased in amount and not purulent, and containing no pathogenic organisms.

Etiology.—It occurs as a sequel to a purulent meningitis, in some cases of internal hydrocephalus, after injury, chronic toxemia, especially alcoholism, acute infectious diseases, delirium tremens, chronic nephritis, and prolonged gastro-intestinal disorders.

Pathology.—The brain and membranes are either injected or pale and edematous. The ventricles are dilated, and the perivascular and pericellular spaces are dilated and contain fluid. The nerve-cells show signs of degeneration.

Symptoms.—The patient complains of headache; cervical rigidity, marked restlessness, and general hyperesthesia are present. Some cases are ushered in with delirium and marked restlessness, which merges into a semicomatose condition, accompanied by a muttering delirium and perhaps flitting delusions and hallucinations.

The tongue is usually coated and dry, the pulse rapid, and there is little or no temperature. Weakness, but not paralysis, of the extremities is present. The pupillary reaction is sluggish, and the patient becomes indifferent to the passage of urine and feces.

As the condition becomes more marked the coma deepens, and the temperature may rise. The skin loses its elasticity, and when picked up and pinched remains elevated for some time; this condition is known as "putty skin." The pulse becomes more rapid and feeble, and bed-sores may develop, the patient dying either from a secondary pneumonia or from cardiac exhaustion. Many cases do not pass into this stage, the mind becoming clearer, the hyperesthesia and restlessness decreasing, and the patient going on to recovery. The cerebrospinal fluid is markedly increased in amount, and it is clear and does not contain pathogenic organisms.

Course.—During the course of an otitis media, or following an injury, prolonged intoxication, etc., symptoms of a meningitis may occur that do not present marked local manifestations, or severe and persistent pyrexia. This condition may clear up within twenty-four hours, or may run a prolonged course, lasting many days, weeks, or even months, during which exacerbations and remissions in the symptoms may manifest themselves.

The **prognosis** varies: those cases in which the symptoms appear as the result of an irritation of the cerebral membranes, in young individuals, usually present a very good prognosis. When the condition occurs in adults, after prolonged intoxication, the prognosis is unfavorable.

Diagnosis.—This condition must be differentiated from other forms of meningitis: in *suppurative meningitis* the temperature is usually higher, there is a leukocytosis, and the cerebrospinal fluid is purulent and contains pathogenic organisms; in *tubercular meningitis* the onset is more insidious, delirium and restlessness are less marked, and local symptoms, pointing to involvement of the base of the brain, are commonly present.

Treatment.—The patient should be placed in bed, in a quiet, well-ventilated, and darkened room. He should be constantly

watched by efficient nurses to prevent injury. Care should be exercised to prevent the development of bed-sores, and attention should be paid to the proper emptying of the bladder and rectum. A careful examination is to be made to remove the cause of irritation if possible. In those cases secondary to an acute otitis media operations upon the ear or mastoid may be followed by immediate disappearance of the symptoms. Lavage of the stomach and free purgation should be promptly instituted in those cases that are caused by gastro-intestinal derangements, and subsequent regulation of the diet should be instituted. Ice-caps should be applied to the head, and mild counterirritation along the course of the spine. The pulse should be sustained with strychnin. Fluids should be given in sufficient quantities. Most important is spinal puncture, the withdrawal of cerebro-spinal fluid being frequently followed by marked improvement in the symptoms; this should be repeated within twenty-four hours if necessary.

CEREBRAL ANEMIA

Etiology.—Anemia of the brain may be produced by the loss of a large amount of blood, by obstruction of the blood-supply to the brain, by cardiac weakness, and by spasmodic contractions of the cerebral arteries. It also occurs in diseased states of the blood, as the result of emotional disturbances, such as excitement, fear, anger, grief, shock, physical pain, childbirth, etc. Neurotic individuals are predisposed to attacks of cerebral anemia.

Symptoms.—The onset of the disease may be either acute or chronic. If the cerebral anemia comes on acutely, there are usually disturbance of consciousness, nausea, vomiting, tinnitus aurium, and vertigo; the individual feels weak, and there is a sense of heaviness, anxiety, or indifference. If the interference with the blood-supply to the brain is very extensive, unconsciousness supervenes, the pupils become dilated, the reflexes are lost, and convulsions may occur.

The face and the mucous membranes are usually pale. The skin feels cold, and may be covered with profuse perspiration. In the milder degrees of cerebral anemia the patient becomes

faint, is depressed, and only partially loses consciousness, everything appearing indistinct before his eyes. He usually falls slowly to the ground. The pulse is generally small and rapid, and breathing is slow, rapid, or irregular. The attacks may last from a few minutes to an hour or more. In those cases in which the cerebral anemia is of more gradual onset and of longer duration, giving rise to a chronic cerebral anemia, the patient complains of symptoms that are usually more marked when he is in an upright position, and less marked when he is lying down.

These patients complain of insomnia or a constant drowsiness and disturbance of the memory. There is a tendency to syncope upon the least exertion, and more or less constant vertigo and tinnitus aurium are present. The patients also complain of discomfort in the head, and at times delirium develops.

Course.—The course is largely dependent upon the etiology. The attack may come on suddenly; in cases due to chronic cerebral anemia there are more or less constant cerebral symptoms, which are aggravated upon exertion, and are more marked with the patient in the erect posture than when he is reclining.

The **prognosis** depends upon the cause. In the less severe cases the outlook for recovery from the attack is good; in the more severe forms, in which convulsions occur and the pupillary reflexes are lost, the prognosis is unfavorable, as the patient may die during the attack or following it. There may be symptoms pointing to cerebral degeneration.

Treatment.—This consists in treating the attack and also the cause. Treatment of the cause resolves itself into making a careful examination to discover the underlying condition. In neurotic individuals, or in those hereditarily predisposed, their reëducation, cautioning them to avoid excitement and to exert self-control, and the general building-up of the bodily condition, are the indications.

Treatment of the attack: The patient should be placed in the horizontal position, with the head as low as possible. Circulation should be stimulated by sprinkling cold water on the skin, applying spirits of ammonia to the nostrils, and administering stimulants by mouth or hypodermically.

CEREBRAL HYPEREMIA

Increase in the amount of blood present in the brain may be the result of interference with the venous circulation, or may be caused by an increased amount of blood being sent to the brain. In the former the hyperemia is passive, and in the latter, active.

Etiology.—Overeating, reflex irritation, increase in the blood-pressure, mental activity, overindulgence in alcoholic liquors, vasomotor instability, disturbances of menstruation, compression of the jugular veins by tumors or enlarged glands, pressure upon the superior vena cava, certain drugs, such as nitroglycerin, amyl nitrite, etc., sunstroke, sudden changes in position and physical exertions, etc., are all among the causes.

Pathology.—The cortex and white matter of the brain present a pinkish appearance, there is an increase in the cerebrospinal fluid, and the blood-vessels and sinuses are overdistended. The veins stand out prominently on the cerebral cortex, and at times pulsation can be noticed in the vessels.

Symptoms.—Cerebral hyperemia may occur either acutely or subacutely, or may develop into a chronic condition. If the attacks are acute, there may be loss of consciousness, mental confusion or delirium, and a mild degree of fever. These symptoms may be accompanied by paralysis or weakness, disturbances of speech, and convulsions; they are generally of brief duration, complete recovery being the rule.

If the attack occurs subacutely, the patient presents a more or less flushed appearance; the conjunctivæ are injected, and he complains of headache, mental dulness or irritability, restlessness or somnolence, and more or less constant tinnitus and vertigo. In chronic hyperemia there are mental heaviness, a tendency to a state of confusion, restlessness, and at times drowsiness or insomnia, inability to perform mental work, headache, and the like. Symptoms due to cerebral hyperemia are usually aggravated when the patient is in the recumbent position, or when changes of position cause the head to be lowered. They generally subside when the patient assumes the upright position.

Diagnosis.—A diagnosis of cerebral hyperemia is less frequently made at present than formerly, since proper examination will generally reveal the presence of the underlying condition that is responsible for the disturbances in the cerebral circulation.

Prognosis.—The prognosis depends entirely upon the cause. In the few cases in which the attacks are not severe and of short duration the prognosis is, as a rule, good.

Treatment.—Cerebral congestion is to be prevented by avoiding excesses of all kinds, such as physical overexertion, etc., and by giving proper attention to the gastro-intestinal tract. Alcoholic liquors should be eschewed, and the patient should lead a life as free from excitement and worry as possible.

Any of the causative conditions that are revealed upon physical examination should be properly treated. During the attack the patient's head should be elevated, and cold applications made to the head. Hot mustard foot-baths are useful. All constricting clothing should be removed, and the room should be cool and well ventilated. If the attack is very severe and persistent, purgatives are to be administered, and in some cases it may be necessary to employ blood-letting. If there is much restlessness, the bromids may be given.

RUPTURE OR OCCLUSION OF THE CEREBRAL ARTERIES

Etiology.—Rupture or occulusion of a cerebral vessel may occur at any age. It may take place during birth, as the result of pressure or injury; during early childhood, following injury, endocarditis, convulsions, etc. The greatest number of cases occur between the fortieth and the sixtieth years. Males are more frequently attacked than females. In some families there is a distinct hereditary predisposition to the occurrence of disease of the cerebral blood-vessels.

Disturbance of the cerebral circulation is due to disease of the blood-vessels, which causes either a weakness of the vessel walls, predisposing to rupture, or decrease in the size of the lumina of vessels, thereby preventing an adequate amount of blood from reaching the cerebral substance; to trauma, rupturing

or compressing a vessel; to some particle that enters or is present in the blood-stream, and is carried into a cerebral vessel, thereby suddenly occluding its lumen; to an abnormal state of the cardiac muscles whereby the force of the circulation is either deficient or increased; to diseased states of the blood itself.

We find, therefore, that the causes of disordered functions of the brain due to a disturbed circulation are principally those that bring about diseased states of the blood-vessels, the heart, and the blood. Among the more common conditions that cause diseased states of the cardiovascular system are syphilis, chronic intoxications, such as alcohol and lead-poisoning, etc.; chronic gastro-intestinal auto-intoxication; excessive ingestion of proteins; old age; heavy work; worry; prolonged constitutional diseases, such as gout, rheumatism, nephritis, infectious diseases, particularly typhoid fever, heredity, etc.

Diseases that bring about or that are accompanied by a myocarditis, cardiac hypertrophy, or endocarditis, or such diseases as leukemia, pernicious anemia, or purpura, all predispose to the occurrence of rupture or occlusion of a cerebral blood-vessel. Among the exciting causes are emotional excitement, physical exertion, trauma, overeating, exhaustion, mental depression, physical weakness, shock, coughing, convulsions, overindulgence in alcoholic liquors, constipation, etc.

Pathology.—As the result of any of the predisposing causes a disturbance of the cerebral circulation may take place, bringing about one of three conditions: (1) A vessel may rupture and result in hemorrhage into or upon the brain; (2) the vessel may be gradually occluded, thereby depriving the brain substance more or less completly of nourishment, and causing a gradual wasting away of the brain substance; (3) a vessel may be suddenly occluded, bringing about an abrupt cutting off of the nourishment of the portion of the brain supplied by that vessel; if this occlusion is permanent, it will result in a softening and shrinkage of that part of the brain supplied by the occluded vessel.

A sclerosis of the peripheral blood-vessels may exist without a similar condition of the cerebral vessels, or a sclerosis of the cerebral vessels may be present without a corresponding sclerosis

of the peripheral vessels. As a rule, however, the condition of the peripheral vessels is a fairly accurate index as to the condition of the cerebral vessels.

Disease of the blood-vessels may result in the formation of new cellular tissue, affecting the endothelial lining of the vessels, causing a gradual obliteration of the lumen; or the middle coat of the vessel may undergo calcareous degeneration, with absorption of the inner layer of the vessel, causing a roughening of the walls. On these roughtened areas fibrin may be deposited, which may gradually occlude the vessel or be carried off as emboli; or the walls of the vessels may undergo degenerative changes, become weakened, and thus permit the formation of small or large aneurysms. Following these degenerative changes, either with or without the production of the aneurysms, there is a marked predisposition to rupture and the occurrence of hemorrhage.

Hemorrhage may occur either on or into the brain substance. When a cerebral vessel ruptures in the membrane, the blood spreads out on the surface of the brain, beneath the arachnoid, filling in the fissures. If the amount is very large, it gradually compresses the brain. If the blood is not evacuated, a clot forms, which may be absorbed, the space that was occupied by the clot being filled by the cerebrospinal fluid. In some cases, especially in older individuals and in alcoholics, the clot becomes organized and adherent to the cerebral membranes. If hemorrhage into the cerebral substance occurs, it destroys the surrounding brain tissue. The amount of blood lost varies from a very small quantity to large amounts; the destruction of the cerebral tissue may be limited to a small area or may involve the entire hemisphere. More than 60 per cent. of cerebral hemorrhages occur as the result of rupture of the lenticulostriate or lenticulothalamic arteries; these arteries are termed by Charcot the hemorrhagic arteries of the brain. Owing to the fact that those on the left side receive the impulses from the heart more directly than those on the right there is a slightly greater tendency for vessels to rupture on the left side of the brain than on the right. A hemorrhage occurring from these vessels destroys, more or less completely, the lenticular nucleus, the optic thalamus or caudate

nucleus, and that portion of the internal capsule in the affected region. If the hemorrhage is very extensive, it may invade the ventricles (Figs. 137, 138).

In about 20 per cent. of cases the hemorrhage occurs on the cortex of the brain, as the result of rupture of miliary aneurysms on the surface vessels of the brain. In a few cases hemorrhage has its origin in a ruptured ventricular vessel, and in others hemorrhage occurs in or on the brain-setm. Only rarely does hemorrhage into the cerebellum occur.

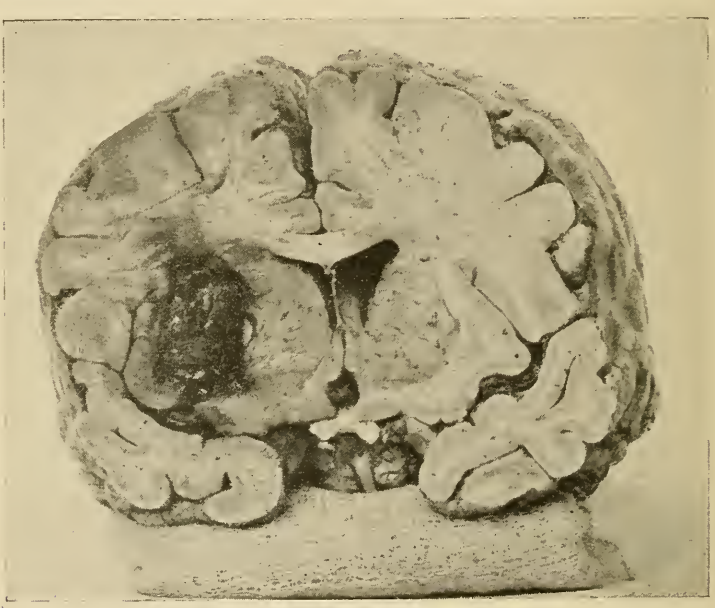


Fig. 137.—Cerebral hemorrhage in internal capsule.

After a hemorrhage into the brain-substance has occurred, a clot forms, which is usually irregular in shape. The brain tissue around the clot is compressed and edematous; gradually the clot shrinks, the edema disappears, and as time goes on there is a gradual softening and absorption of the clot and of the disintegrated brain tissue. Eventually there remains an area of connective tissue which may be pigmented. In a few cases a cyst forms during the course of the absorption of the clot.

After a hemorrhage has taken place the clot begins to lose its

reddish color in from the second to the fifth week, and in the eighth to the twelfth week it becomes yellowish. Connective-tissue formation is well established within six months, and contraction of the connective tissue continues until the end of the second year.

As the result of destruction of the brain tissue secondary degeneration begins at once, and by the end of the second week this degeneration can be detected at remote points from the original lesion. In such diseases as purpura, pernicious anemia,

Fig. 138.—Brain, showing extensive hemorrhage into ventricle.

etc., there may be extravasation of blood without apparent rupture of vessels. In these diseases the blood collects about the vessels and infiltrates and compresses the brain tissue in the vicinity of the vessels, bringing about interference with or complete loss of the functions of the affected portions of the brain.

The lumen of the cerebral vessel may be gradually occluded as the result of thickening and proliferation of the endothelial lining, leading to gradual interference with the blood-supply of the part of the brain nourished by the diseased vessel; or the

lumen of a vessel may be suddenly occluded by an embolus coming from some distant point, such as the heart, other blood-vessels, the lungs, etc., or by a clot forming upon the roughened wall of a diseased vessel. In this condition there is a sudden interference with the blood-supply of the part of the brain nourished by the occluded vessel; usually this is at first very extensive. In addition to the interference with the blood-supply there is also, as a rule, an edema of the surrounding cerebral tissues.

Owing to the fact that there is a free anastomosis of the vessels upon the surface of the brain, shortly after one of these vessels is occluded a free collateral circulation takes place, and the cerebral tissues again receive their blood-supply. The vessels that penetrate the brain substance are all terminal vessels; if, therefore, they become occluded, the portion of the brain supplied by them is permanently deprived of its nourishment, and consequently undergoes degenerative changes. As the result of the deprivation of blood the cerebral tissues undergo softening, with degeneration of the cellular constituents. At times there is an extravasation of blood into the softened area, when the condition is known as red softening.

Following the degenerative changes the softened tissue undergoes liquefaction, or becomes organized by connective-tissue growth: in the former case a cyst is produced with smooth walls (Fig. 139), and in the latter case there is left connective tissue or a scar in the cerebral tissue. The size of the area of softening depends upon the area of cerebral tissue supplied by the occluded vessel. In those cases in which the occluding agent contains pathogenic microörganisms there is added to the pathologic changes that result from interference with the blood-supply the evidence of an acute encephalitis of more or less limited extent.

Owing to the fact that the circulation from the heart is more direct to the left side of the brain and to the middle cerebral artery, 60 per cent. of emboli are carried into the left middle cerebral artery or its branches.

Symptoms.—The symptoms of disturbance of the blood-supply of the brain may in most cases be divided into two periods —the first period, during which there is a partial, temporary, or

intermittent disturbance of the blood-supply; the second period, during which there is a more or less sudden complete interference with the supply of blood to a definite area in the brain, or there occurs a rupture of a vessel with the extravasation of blood.

The symptoms occurring in the first period are called the premonitory symptoms: they are the general symptoms and signs of cardiovascular diseases, such as rapid or irregular

Fig. 139.—Cystic formation following vascular occlusion.

pulse, high or low blood-pressure, valvular murmurs, thickened and tortuous vessels, disturbance of the heart sounds, evidence of chronic interstitial nephritis, gastric disturbances, etc. With or without these there usually occur symptoms pointing to disturbance of the cerebral circulation. Of these, the commonest is *headache*, which is generally of a dull character, and grows worse on exertion, excitement, or by anything that tends to weaken the individual. These headaches are not usually continuous, but come on in attacks that vary in frequency and

severity. Attacks of *vertigo* appear, which vary in severity and come on at irregular intervals, usually after exertion, emotional disturbance, excitement, etc. *Paresthesia:* Nearly all patients with vascular disease of the brain at some time or other complain of numbness, tingling, formication, etc; these abnormal sensations may be limited to one entire limb or to one-half of the body; on the other hand, these sensations may affect the terminal portions of all four extremities.

Disturbances of speech, the patient finding it difficult to express his thoughts in words, occur at intervals, but usually rapidly disappear. Many of these individuals will also complain of loss of memory, especially for recent events; difficulty in fixing the attention on important matters, there being a constant tendency for the mind to wander; difficulty in thinking, abnormal irritability, and a tendency to become emotional.

In the more advanced cases attacks of confusion or stupor may occur. Disturbance of sleep may also arise, the patient being very restless on going to bed and unable to sleep until toward morning; other patients go to sleep almost immediately upon retiring, but awaken after a few hours and find it impossible to go to sleep again. Others may be drowsy and sleep almost the entire day, but will be unable to sleep during the night. Tinnitus aurium, visual disturbances, etc., may also occur.

In some patients there will be attacks of weakness, or even paralysis, affecting one limb or one side of the body; these attacks are of short duration. These premonitory symptoms may occur over a period of many years before evidence pointing to permanent occlusion or rupture manifests itself. On the other hand, in some cases, without the appearance of any of these premonitory signs, there will be symptoms pointing to rupture or occlusion of a vessel. This condition is, of course, in many cases easily explainable. In some cases an embolus is carried from the heart or some large vessel to a cerebral vessel, which it suddenly occludes. This may occur in one who has previously had no disease of the cerebral blood-vessels. Miliary aneurysms of the cerebral vessels may give rise to no symptoms, and yet rupture, when for some reason or other there is an increase in the blood-pressure.

The clinical state that occurs as the result of an occlusion or rupture of a cerebral vessel is termed **apoplexy.** An apoplectic attack usually comes on more or less suddenly, and is characterized by evidences of the loss of function of some portion of the brain. As a rule, it is accompanied by considerable mental shock. In some cases the apoplectic attack may come on slowly, covering a period of from a few minutes to several days.

Symptoms of the Attack.—In about half the cases loss of consciousness occurs simultaneously with the attack; the degree and duration of the unconsciousness serve as a fairly accurate index of the severity of the seizure.

General Constitutional Symptoms.—There are two types of patients: in the one the pulse is slow and regular, the blood-pressure ranging from 160 to 250; the face is flushed or cyanotic; the breathing is stertorous and irregular; the conjunctivæ are injected, and the temperature ranges from the normal to 104° or 105° F. Edema of the lungs may occur, and the patient die within a few hours or days, either from cardiac exhaustion or from pulmonary edema.

In the second type of cases disturbances of consciousness are not so marked, and in some they are very slight; the pulse is more or less rapid, and perhaps irregular; the blood-pressure is low; the face is pale; breathing is irregular or of the Cheyne-Stokes variety, and there is no temperature.

In some cases at the onset of the attack there is a relaxation of the rectal and vesical sphincters, with an involuntary evacuation of the contents of the bladder and rectum. In those cases attended by coma the patient falls, and cannot be aroused; he is motionless and insensible, and the face is usually drawn to one side, with a dribbling of saliva upon the opposite side. There is a loss of the skin reflexes, and later on increased tendon-reflexes. Automatic movements may occur on the non-paralyzed side.

Convulsive movements may usher in the attack or may occur later on. Disturbance of consciousness may last for a few hours, and be followed by a state of mental torpidity and confusion that may last several days or weeks. The comatose state is apt to occur in hemorrhage of the deeper structures of

the brain, or in occlusion of large vessels. Lesions in the cerebellum and brain-stem are less likely to be accompanied by disturbances of consciousness.

Within from twenty-four to forty-eight hours after the attack it is usually possible to determine the location, the extent, and the character of the lesion.

Location of the Lesion.—In lesions on the surface of the brain the symptoms are generally of slower onset than are those occurring in the deeper structures; they manifest themselves first by symptoms of irritation of the cortex, which are later replaced by those of paralysis. Symptoms due to lesions on the surface of the brain are usually much more circumscribed than are those due to involvement of the deeper structures.

Symptoms pointing to involvement of the anterior part of the frontal lobe are variable and indefinite: there may be emotional disturbance, a tendency to cry and laugh without cause, changes in character, disturbance of memory, confusion, intellectual weakness, irritability, attacks of excitement or depression, etc. If the third frontal convolution or its projection pathways in the left hemisphere in right-handed individuals, and the right hemisphere in left-handed individuals, is involved, there occurs a disturbance of speech known as aphasia. In this condition the individual knows what he wishes to say and recognizes objects, but is unable to express himself or to call objects by their names. In some instances the patient may be able to utter a few meaningless sounds or even words.

If the precentral convolution or its projection pathways are involved, disturbance of motility occurs. According to the severity of the lesion, there occurs either a weakness or a paralysis; this manifests itself as a disturbance of voluntary movements. The more highly specialized and the most difficult acquired movements are those which are always most seriously affected. If the lesion is on the cortex, the paralysis or weakness may be of very limited extent, involving the facial movements or those of an arm or a leg; or, if more extensive, perhaps the face, or the face and arm, or the arm and the leg, etc., may be affected.

An apoplectic seizure due to a surface lesion in this region

may be ushered in by convulsive movements that are at first limited in extent, but later may become extensive and be followed by paralysis, etc. If the lesion is beneath the surface, that is, in the projection paths from the precentral convolution, the paralysis or weakness is generally much more extensive, particularly if the lesion involves the anterior two-thirds of the posterior limb of the internal capsule. In this position a very small lesion brings about a paralysis of the arm, leg, face, and trunk of one side of the body. This condition is known as hemiplegia.

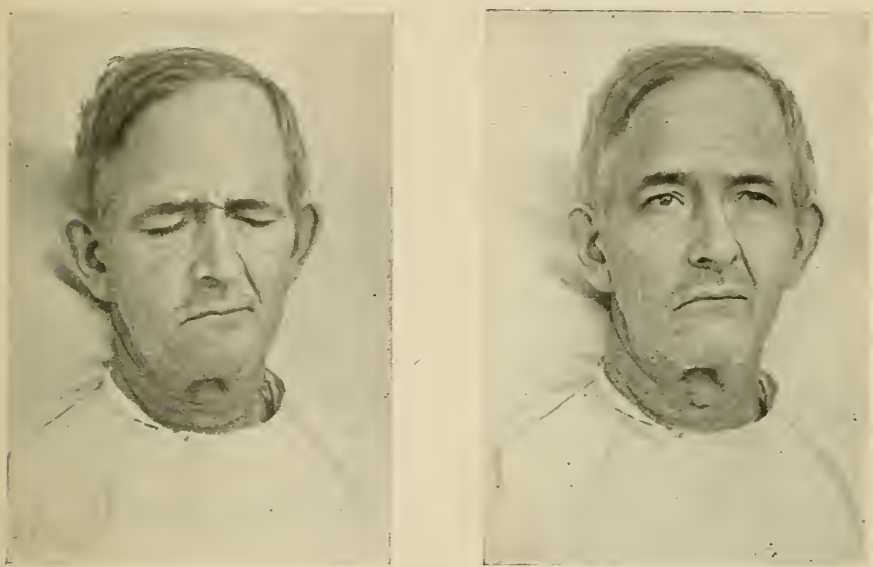


Fig. 140.—Facial paralysis following apoplectic stroke in patient with complete aphasia.

Immediately after an attack there is generally complete relaxation of all the muscles. Within a few hours, even if the patient does not move the non-paralyzed side, a greater relaxation on one side is observed, together with a drawing of the face, an elevation of the corner of the mouth on the non-paralyzed side (Fig. 140), with dribbling of saliva on the paralyzed side, difficulty in swallowing, etc. Within a short time an increased tonicity and a tendency to contractures of the muscles on the paralyzed side are noticed. This is more marked in cases in which the lesions are subcortical than in those in which the

cortex is affected. After an attack the head and eyes are usually turned away from the paralyzed side; that is, they deviate toward the site of the lesion.

During the comatose or semiconscious state automatic instinctive movements may occur on the non-paralyzed side. Convulsive movements occurring after an apoplectic seizure usually indicate rupture of a vessel and hemorrhage into the ventricles. Apoplectic seizures that are ushered in with sudden, severe, generalized convulsive attacks generally indicate a primary involvement of the ventricles. If recovery is to take place, within a short time after an apoplectic seizure there is a gradual return of some of the movements of the paralyzed side; the first to occur are the grosser movements of the leg and arms. In some there is almost complete recovery, even of the finer movements of the fingers and toes; in most cases in which recovery does take place there remains some clumsiness in affecting the finer movements of the fingers and hands.

Fig. 141.—Posture in hemiplegia.

The gait of those who have sustained an attack of hemiplegia and who still live is more or less characteristic: the patient walks with the trunk thrown forward; the shoulder on the paralyzed side is lower than that on the non-paralyzed side; the arm is adducted on the trunk; the forearm is flexed on the arm; the hand is pronated and flexed at the wrist, and the fingers are flexed (Fig. 141).

The paralyzed leg is held stiff. The foot may be turned outward or inward—usually outward. When the patient walks, he does not bend the knee, but swings the foot outward in the arc of a circle, usually drawing the ball and the inner aspect of the foot along the ground. The efforts made when walking are frequently attended by an increase in the contracture of the arm, and sometimes of the face. Not infrequently, after the occurrence of a hemiplegic attack, associated and athetoid movements in the paralyzed limbs take place.

The Reflexes.—Immediately after an apoplectic seizure, if the patient is unconscious, the reflexes are usually lost, there is a loss of the pupillary reaction to light, and there is relaxation of the vesical and rectal sphincters, with an emptying of the contents of bladder and rectum. Within a few hours, or at the most days, if a fatal termination is not imminent, there is usually a return of the tendon reflexes, which later become markedly increased. Ankle-clonus and the Babinski sign often occur on the side of the paralysis within a few days after an attack. In the less serious attacks, in which there has not been so much destruction of tissue, the deep reflexes return rapidly and soon become increased, but the skin reflexes on the paralyzed side are permanently lost. The pupillary reflexes gradually return as the unconsciousness begins to subside. With the return of the tendon reflexes there is also a return of the reflex control of the rectum and bladder. As there is usually retention of urine and feces, it may be necessary to resort to catheterization to empty the bladder, and to enema to clear the rectum. At other times there may be involuntary evacuation of bowel and bladder contents.

If the lesion involves the anterior part of the parietal lobe, transient motor symptoms will occur, and if the lesion is on the surface of the brain, paresthesia may occur upon the opposite side, to be followed later by anesthesia, which usually almost completely disappears. If the lesion is in the projection paths from the parietal lobe, there also occurs paresthesia, followed by anesthesia, and there are generally added to these disturbances of intellect, due to destruction of the association

pathways. The sensory disturbances are not permanent as a rule.

If the lesion affects the angular and supramarginal gyrus, astereognosis occurs. If the lesion involves the temporal lobe, there may be disturbance of smell and of hearing; if the lesion involves the lateral aspect of the occipital lobe, alexia occurs; if the lesion involves the median aspect of the occipital lobe or its projection pathways, bilateral hemianopsia occurs, with retention of the pupillary reflex. In those cases in which the lesion occurs in the cerebellum,—and these are certainly uncommon,—there may or may not be a disturbance of consciousness. If the consciousness is not disturbed, or if lost, after it has been recovered, the patient experiences intense vertigo, double vision, nystagmus, attacks of vomiting, and severe prostration. There is usually marked incoördination of movement, and the gait is staggering. The patient may recover from all these symptoms or the vertigo and incoördination may remain.

If the lesion involves the crura, pons, or medulla, a characteristic symptom-complex presents itself: if the lesion affects the crura, involving only one side, there occurs a paralysis of the muscles supplied by the third and fourth nerves on the side of the lesion, with a spastic paralysis on the opposite side of the body, including the face, tongue, upper extremity, trunk, and leg. Lesions in the pons give rise to great restlessness and paralysis of one or both sides of the body; if the lesion involves only one side of the pons, there will be a paralysis of the fifth nerve on one side, with paralysis on the opposite side of the body. In pontile lesions the pupils are usually contracted, and the patient, if not unconscious, is very emotional. If the lesion involves the lower part of the pons or the upper part of the medulla, there occurs a paralysis of the muscles supplied by the sixth and seventh nerves on the side of the lesion and the opposite side of the body. If the lesion in this region involves both sides of the brain-stem, there may be a paralysis of the muscles supplied by the sixth and seventh nerves on both sides, with or without paralysis on one or both sides of the body below.

Vascular lesions below the upper part of the medulla, if they are at all extensive, cause death more or less promptly. Very small lesions may exist, and these may give rise to symptoms pointing to involvement of the nuclei of the twelfth, ninth, or tenth nerves, resulting in paralysis of the tongue, and difficulty in swallowing and in talking. If the lesion affecting the brain-stem involves the deeper structures, there may be no paralysis, but disturbance of sensation and of coördination may accompany the cranial nerve paralysis. Paralysis of the cranial nerves that occurs directly as the result of the lesion in the brain stem is always of the flaccid type, whereas paralysis of the parts of the body below the lesion is of the spastic type.

Course.—Diseased conditions of the brain due to vascular disturbance may be acute in onset, but usually occur only after manifestations of a disturbed circulation have existed for a considerable time. Symptoms pointing to vascular disease of the brain may exist for many years, or, on the other hand, rupture or occlusion of a vessel may take place without any of the premonitory symptoms. When an apoplectic attack has occurred, the patient may or may not become unconscious,—the period of unconsciousness may last from a few minutes to several days. The patient may die within a few hours after the attack, or may gradually emerge from the comatose state. If the patient has been unconscious and emerges from this state, there is usually a period of mental heaviness and confusion lasting several days or weeks; the patient may die during this time or gradually regain consciousness.

As soon as consciousness recurs, or in those patients who have not been unconscious, the breathing again becomes normal, the ability to swallow is regained, and control of rectal and vesical reflexes is reëstablished. Those patients who are not aphasic begin to express their desires by speech, at first very indistinctly, but within a few days fairly well. The paralysis, which at first completely affects all movements of one side, may gradually improve, the grosser movements of the extremities being the first to return. In some cases the paralysis may disappear almost entirely, whereas in other cases very little improvement may be noticed.

Contractures, associated, automatic, or athetoid movements may appear.

Prognosis.—The prognosis in apoplectic attacks is always grave, owing to the fact that not alone is the attack itself a serious occurrence, but because of the tendency for subsequent attacks to occur. Those cases ushered in with violent convulsive seizures, followed by unconsciousness, generally die within three or four days. In those patients who remain unconscious for over four days, and in whom there is some elevation of temperature with a high blood-pressure, the outlook is very serious, as death from pulmonary involvement or exhaustion is very prone to ensue. When the symptoms point to extensive involvement of the brain-stem the patient almost always dies within three days. In those cases in which there is less disturbance of consciousness, or in those patients who rapidly regain consciousness, and the examination of the heart and lungs does not lead to apprehension, recovery almost always ensues. Subsequent attacks may occur—sometimes within twenty-four hours. Following a seizure some individuals remain very emotional, weeping or laughing without adequate cause; others show disturbance of memory, marked irritability, mental confusion, etc.

Recovery of the motor functions upon the affected side may take place in whole or in part: usually the face recovers almost entirely, and very little deviation of the tongue remains; voluntary power of the upper and lower extremities, particularly of the finer movements, may, however, be permanently lost; as a rule, less voluntary control is recovered in the arm than in the leg.

The paralysis of the affected parts is of a spastic nature, contractures and abnormal movements developing in some cases, whereas in others voluntary power may be regained to a considerable extent, disturbance of sensation being the only permanent symptom. Hemianopsia is usually permanent.

Diagnosis.—The diagnosis of vascular disease of the brain is made from the evidence of general cardiovascular disease and from the occurrence of symptoms pointing toward interference with the cerebral circulation, such as paresthesia, limited to

one arm or one side of the body, in the absence of local causes; vertigo, headache, disturbances of sleep, disorders of speech, emotional disturbances, transient attacks of weakness limited to one side of the body, etc. If an apoplectic attack occurs, it must be differentiated from an attack of *syncope:* in the latter condition the patient's face is usually pale, the respiration is shallow, the cardiac sounds are distant and weak, the pupils are equally dilated, and there is retention of the knee reflexes, with rapid recovery following the administration of stimulants. From an *epileptic attack* an apoplectic seizure is distinguished by the fact that in the former there is generally a history and local evidence of previous epileptic seizures, such as scars upon the tongue or upon the person; moreover, epileptic seizures occur more frequently in youth, whereas apoplectic attacks occur later in life. In epilepsy signs of cardiovascular disease are generally absent, whereas in apoplexy they are usually present. The coma following an epileptic seizure is short, and when the patient has regained consciousness there is an absence of symptoms pointing to a lesion of the brain.

An apoplectic attack is distinguished from a comatose state the result of intoxication from alcohol, opium, etc., by the fact that in the latter the individual can usually be roused; there is also an absence of symptoms pointing to local brain involvement, and practically never is there a loss of the pupillary reflexes. Owing to the fact that apoplectic attacks may occur in persons who are under the influence of alcohol it is important that in examining these individuals search be made for symptoms of localized brain involvement. It is at times very difficult to distinguish between an apoplectic attack and uremic coma. When no history is obtainable, it is only by the presence of symptoms pointing to localized involvement of the brain that one is enabled to distinguish between a vascular disturbance of the brain and a toxic condition. It must constantly be borne in mind that apoplectic attacks are of somewhat common occurrence in persons who are affected with a chronic interstitial nephritis. It is important, but at times difficult or almost impossible, to differentiate between apoplectic attacks

caused by hemorrhage, thrombosis, or embolus. The following table is useful in making the differentiation:

	Hemorrhage.	Thrombosis.	Embolus.
Age:	Occurs most frequently before the seventh year and after the fortieth year.	Occurs most frequently in old age, in young adults who have had syphilis, or in individuals who have become very much weakened as the result of some prolonged exhausting disease.	Occurs at any age.
Predisposing causes:	Heredity is a marked cause. Arteriosclerosis with high blood-pressure.	Heredity an uncommon cause. Arteriosclerosis with low blood-pressure.	Heredity has no influence. Endocarditis, calcareous degeneration, with roughened vascular walls, etc.
Exciting causes:	Excitement, overexertion, trauma, convulsions, etc.	Sleep, diseased conditions bringing about a low blood-pressure, depression, extensive hemorrhage, etc.	Excitement, overexertion, injury, infectious diseases, convulsions, etc.
Onset:	Prodromal symptoms may or may not exist. Attacks usually very sudden.	Prodromal symptoms common. More or less gradual, frequently taking several hours for attack to attain its maximum degree.	Prodromal symptoms usually do not exist. Attack sudden.
Appearance:	Face usually congested; conjunctivæ injected; lips cyanotic.	Face usually pale.	Face may be either congested or pale.
Coma:	Common; usually sudden and complete.	Uncommon; more gradual in onset and usually not well marked.	Uncommon, but when it occurs, is of sudden onset, and usually rapidly recovered from.
Respiration:	Usually sterterous, irregular, or of the Cheyne-Stokes variety.	Frequently no disturbance; irregular.	Generally no disturbance.
Pulse:	Usually slow, full, and bounding.	Usually weak, soft, and rapid.	May be either slow or rapid, but is usually full and well sustained.
Distribution of motor symptoms:	Hemiplegia usually complete, followed by improvement.	More limited, but frequently becomes more extensive.	May be limited or extensive, often with rapid improvement.
Temperature:	At first reduced and later elevated.	No changes.	No changes.
Eyes:	Pupils unequal; pupils fail to react to light. Eyes generally turned away from the side of the paralysis.	Equal. React, unless coma is present. Usually no deviation.	Equal. React unless coma is present. Usually no deviation.
Blood:	A rapid increase in the leukocytes.	No increase.	No increase.
Vasomotor symptoms:	Edema, cyanosis, and increased sweating common.	Uncommon.	Uncommon.
Urine:	Frequently increased; may contain albumin and sugar.	No changes over previous condition noticed.	No changes over previous condition noticed.

Treatment.—The treatment of diseases of the brain due to disturbance of the cerebral circulation, to be efficient, must be directed toward the prevention and more or less permanent relief of the underlying cardiovascular diseases. Proper attention must be given to the diet, and alcoholic beverages avoided. The use of tobacco, coffee, etc., is to be limited. The bowels should be kept well open, and an adequate amount of water should be taken. A moderate amount of exercise, such as walking, horseback riding, golf, etc., should be indulged in, and all overexertion and excitement should be avoided. Very hot or very cold baths are not to be indulged in, but tepid or cool sponges are to be recommended. All acute disease should receive prompt and careful attention. Overeating, exposure to inclement weather,—either too hot or too cold,—overwork, etc., are all harmful. If the patient is debilitated, anemic, and weak, rest, together with proper food and tonics, is to be prescribed. Such diseases as syphilis and nephritis should receive the proper treatment. In all cases in which arteriosclerosis is present the administration of potassium iodid in 5-grain doses, given at intervals over a prolonged period, is of benefit, provided it does not disagree with the patient.

The treatment of an apoplectic attack is dependent upon the cause. The patient should be placed in a comfortable reclining position, with the clothing loosened, and, above all, be kept quiet. He should be moved as little as possible. In my experience the pulse-rate and the blood-pressure are usually the safest guides as to the administration of drugs and as to further treatment: in those cases in which the blood-pressure is high, that is, from 160 to 200 or more, and in which the pulse ranges from 60 to 110, it is advisable to place ice-caps upon the head and to administer a rapidly acting purgative, such as croton oil. If the patient is very restless, small doses of morphin, or if he is not unconscious, sodium bromid, may be given. The pulse-rate and the blood-pressure must be carefully watched: if the pulse becomes slower and the blood-pressure rises, this is an indication of increased intracranial pressure, and it may be necessary to open the cranium in order to save the patient's

life. This should, of course, be promptly done if a cortical hemorrhage is suspected. In those cases in which it is believed that the hemorrhage is deeper in the brain substance an operation may save the patient's life, but it cannot, of course, restore the destroyed cerebral tissue. In those cases in which the face is cyanosed and flushed, the pulse slow, full and bounding, and the blood-pressure high, if the patient is not promptly benefited by free purgation and the application of ice-caps to the head, venesection may prove useful. If the patient is unconscious, care should be taken that he is placed in such a position as to render his breathing as easy as possible. He should be turned carefully from side to side, always permitting him to rest longest on the non-paralyzed side. Attention should be given to the bowels and bladder, and care should be exercised to prevent the development of bed-sores. Food should be withheld. The mouth should be kept clean, and small quantities of water should be frequently administered.

In those cases in which the face is pale, the blood-pressure low, and the pulse more or less rapid, the patient should be placed in bed, with his head slightly, if at all, elevated. His position should be frequently but carefully changed from side to side, and cardiac stimulants should be administered. These cases frequently do well on brandy given in small doses, strychnin, aromatic spirits of ammonia, camphor, etc. Strophanthin hypodermically, followed by the digitalis preparations, is of great value at times in those cases in which the cardiac muscle is responsible for the disturbed cerebral circulation. As soon as possible the patient should receive a nutritious but light diet, and tonics be administered. When the exciting cause is believed to be an embolus, providing there are no cardiac contraindications, nitroglycerin and strychnin are to be given, so that the embolus will be pushed as far forward as possible into the occluded vessel.

The less severe cases, in which there is no disturbance of consciousness, should receive the same careful attention that is given the most severe cases.

The diet should be regulated, and fluids given in abundance. The position of the patient should be changed from time to

time, and he should be made as comfortable as possible. The bowels are to be regulated, and proper attention given to the skin.

At the end of ten days or two weeks in the mild cases, and in from four to six or eight weeks in the more severe cases, the patient may be allowed to sit in a chair, and later be permitted to make attempts at standing. While he is making these attempts at standing and sitting the patient should be supported, and not allowed to overexert himself.

About two weeks after an attack massage of the affected parts should be begun, passive movements carried out, and the faradic current applied. By means of the electric current, massage, and passive movements contractures should be prevented as far as possible. Gradually, as the voluntary movements return, they should be encouraged. In those cases in which cortical hemorrhage is diagnosed, prompt surgical intervention is justifiable. Some cases of subcortical hemorrhage in which there is evidence pointing to a fatal termination may be saved temporarily at least by surgical intervention.

In those patients who suffer with sensory disturbances improvement usually follows the application of the faradic current. Aphasia is to be treated by reëducation; the mental and emotional symptoms are generally not benefited by treatment, but these patients should have agreeable, pleasant, and quiet surroundings, and be spared all excitement and mental distress.

THROMBOSIS OF THE CEREBRAL SINUSES AND VEINS

Etiology.—Thrombosis of the cerebral sinuses or veins may occur as a primary affection in individuals who are markedly debilitated as the result of prolonged exhausting disease, in general cachectic conditions, cardiac weakness, after very severe diarrhea, tuberculosis, malignant growths, etc. This condition occurs more frequently in very young or very old individuals. A primary thrombosis may also occur as the result of pressure on veins or a sinus resulting from a tumor or

an injury. It may also occur as a concomitant of inflammation of the venous walls due to extension of an inflammatory process, or may be secondary to a septic process elsewhere. In these cases the thrombus, if infected, contains bacteria. The commonest causes of infective sinus thrombosis are otitis media, chronic nasal or accessory nasal sinus disease, septic traumatic conditions of the face, scalp, and cranial and facial bones, erysipelas, carbuncles or abscesses, meningitis, etc. Probably the most frequent cause of infected sinus thrombosis is otitis media or mastoiditis.

Pathology.—A thrombus may be confined to one sinus, or to even a part of it, or, by gradual extension, several sinuses may be invaded. When the entire lumen of a cortical vein is occluded, the portion of the brain drained by this vein is generally the seat of edema and softening. The lumen of the vein or sinus is usually occluded by a soft clot, which is adherent to its walls. In those cases that have existed for some time the clot may undergo organization. When the thrombus becomes infected, the clot usually breaks down, the vascular walls become rough, and there is an extension of the infection into the immediate neighborhood. The clot may extend, and the internal jugular vein is often invaded. If the particles are carried from the infected clot, a general pyemic condition may result, the septic emboli being carried to the various organs of the body.

As the result of occlusion of the sinuses there occurs a congestion of the brain in the areas drained by the sinus, and frequently there is red softening in these areas.

Symptoms.—The symptoms of a primary non-septic thrombosis are those of a localized edema and interference or loss of function of a portion of the brain. This condition usually follows a prolonged exhausting illness. The superior longitudinal sinus is the one that is most frequently affected. When this sinus is occluded, there generally results edema of the scalp and epistaxis, and frequently convulsions, which may be limited in extent, and be accompanied by weakness or paralysis of the lower extremities.

The presence of a primary thrombosis may be suspected in

individuals in whom an exhausting disease exists, and when an edema of the face or scalp is followed by convulsions, symptoms of meningeal irritation, weakness, or paralysis.

The symptoms of an infective thrombosis are of two varieties, —the systemic and the local. The systemic symptoms are those of a general systemic infection; they include: Headache, which may be very intense; gastro-intestinal disturbances, such as vomiting and loss of appetite; marked elevation alternating with depression of temperature; a pulse usually rapid and small; chills, profuse perspiration, leukocytosis, a dry, coated tongue, etc. To these symptoms may be added others pointing toward involvement of any of the organs of the body. If septic emboli are carried to the lungs, symptoms occur pointing to involvement of these organs; if the abdominal organs are involved, the symptoms point toward the viscera. In some cases there may be symptoms pointing toward meningeal irritation, and a diffuse leptomeningitis may occur.

The localized symptoms are due to local circulatory disturbances and to a localized involvement of the brain. These symptoms depend upon the site of the pathologic lesion.

When the cavernous sinus is involved, the orbital contents become edematous, and exophthalmos, sometimes bilateral, occurs. The eyelids of one or both sides are swollen, the edema sometimes extending to the side of the face. The optic disc is congested, and the retinal veins are distended. If pressure is exerted on the third, fourth, and sixth nerves, evidence of interference with their functions exists. The involvement of this sinus is usually secondary to a purulent disease of the orbit, sphenoidal sinuses, erysipelas of the face, ulcerative conditions of the throat and mouth, and extension forward of septic conditions of the lateral and petrosal sinuses.

Local Symptoms of Infective Thrombosis of the Longitudinal Sinus.—This sinus is very rarely affected. The involvement may be secondary to purulent diseases of the nose, erysipelas of the scalp, purulent conditions of the cranial bones of the vertex of the skull, etc. The local evidences of occlusion are edema of the scalp, nose-bleed, and sometimes symptoms pointing toward an edema of the vertical portion of the brain.

Infective Lateral Sinus Thrombosis.—The lateral sinus is the one most frequently affected. The most common cause of an infective disease of this sinus is a septic condition of the middle ear. It may also arise secondary to disease of the throat, the infection traveling by way of the Eustachian tubes, from fracture of the base of the skull, from extension of septic thrombosis in other sinuses, and it may also be secondary to infections about the occipital and mastoid regions.

Local Symptoms.—As a rule there is a circumscribed edema over the mastoid. Sometimes a brawny hardness and edema in the superior cervical triangle occurs, and should the inflammation extend into the internal jugular, which it frequently does, local tenderness is found over the upper portion of this vessel. At times the internal jugular vein may be palpated as a firm, cord-like structure. The head is usually inclined to the affected side and the lymphatic glands in the neck are frequently enlarged. In some cases pressure is exerted in the jugular foramen upon the pneumogastric, spinal accessory, and glossopharyngeal nerves, producing respiratory, laryngeal, and cardiac disturbances, difficulty in swallowing, and weakness or paralysis of the sternomastoid and trapezius muscles. To these symptoms are generally added evidences of disturbed cerebral circulation and disordered cerebral functions. Generally headache and vomiting are marked, and there is a tendency toward somnolence, which may deepen into coma. Delirium often occurs as an early symptom. Later the infection may extend into the temporosphenoidal lobe, forming an abscess in this region, or it may extend inward into the cerebellum, where it gives rise to an abscess, or the dura may become affected and local septic meningitis develop.

Course.—A primary thrombosis of the cerebral sinus is always preceded by some disease that brings about a marked lowering of the vitality of the individual and a slowing of the blood-current. More or less suddenly symptoms pointing to occlusion of the cerebral sinus or cerebral veins appear. A secondary or septic thrombosis is likewise always preceded by some disease or condition that causes the formation of an infected clot in a cerebral sinus or a cerebral vein. The rapidity

and severity of the symptoms depend upon the virulence of the infecting organism. Usually to the symptoms of the peëxisting condition there are added, more or less suddenly, evidence of occlusion of a cerebral sinus or vein. Some cases run a course to a fatal termination within a few days, whereas others may last for weeks or months.

Prognosis.—The prognosis of a primary cerebral thrombosis depends upon the nature of the initial illness. If the causative disease can be controlled, there is a tendency for reabsorption of the clot to take place, and the only permanent symptoms will be those that arise from destruction of the cerebral tissue. The prognosis of septic or secondary thrombosis is extremely serious: in only a few cases, limited to affections of the lateral sinus, is recovery to be expected. The appearance of an encephalitis, a general septicemia, or a meningitis makes the outlook almost hopeless.

Diagnosis.—The diagnosis of a primary cerebral venous or sinus thrombosis is made when, in the course of some preëxisting chronic debilitating disease, there occurs evidence pointing to disturbance of the functions of the brain, to which is added a localized edema of the face or scalp. In the absence of edema the diagnosis is made only with difficulty. If, however, in the course of a chronic debilitating disease symptoms pointing toward cerebral irritation occur, such as convulsions or a loss of cerebral functions, *e. g.*, bilateral weakness or paralysis, a thrombosis of the superior longitudinal sinus should be suspected. The diagnosis of a secondary or septic thrombosis of the cerebral vein or sinuses is made from the presence of a primary infectious condition, to which are added the symptoms of a general systemic infection and evidence pointing to interference with the circulation of blood through one of the cerebral sinuses, together with symptoms indicating interference with the functions of a localized portion of the brain.

Treatment.—The most important indication is the prompt and efficient treatment of all conditions and diseases that are likely to be complicated by a cerebral sinus thrombosis. If sinus thrombosis has occurred, the underlying disease should receive prompt and skilful treatment. The local focus of in-

fection demands immediate surgical attention. Pain and discomfort may be relieved by the local application of cold or heat to the head. The gastro-intestinal symptoms should receive attention, and free purgation induced. If the patient is restless, bromids or morphin may be given; for the temperature, cold sponges or packs may be applied. General supportive measures are indicated, and the room in which the patient lies should be quiet and well ventilated.

As a rule, in infective thrombosis there is only one mode of treatment, and that is surgical. The infected sinus should be exposed, and the clot removed. The operation must, of course, be undertaken only by one having the proper experience, and every precaution taken to prevent the spread of the infected material. This is best accomplished by ligating the internal jugular vein before operating upon the lateral sinus.

ACUTE NON-SUPPURATIVE HEMORRHAGIC ENCEPHALITIS

Etiology.—An acute non-suppurative inflammation of the brain may occur as the result of an acute infectious disease. Cases of this kind have been known to occur during an influenza epidemic; they may also occur during the course of various infectious diseases, such as scarlet fever, measles, whooping-cough, mumps, and pneumonia, and may complicate erysipelas, ulcerative endocarditis, septicemia, ptomain-poisoning, chronic alcoholism, trauma, otitis media, chronic auto-intoxication, etc.

Pathology.—The inflammatory process is frequently limited in extent. It usually occurs in circumscribed areas, and may involve the cortex and the central ganglia. It may be limited to the gray matter of the aqueduct of Sylvius, in the pons or medulla, and is sometimes limited to the distribution of one artery or to symmetrically situated regions of the brain. The cerebral tissue in the affected area presents a hyperemic appearance, with numerous small punctate hemorrhages, and is somewhat swollen and more moist than normal.

Microscopically, the small arteries and capillaries are seen to

be dilated. Rupture of the vessel walls occurs, and there is an infiltration of white and red cells. The cells of the cerebral substance show evidence of irritation and disintegration.

The distribution and the permanence of the symptoms depend upon the extent and severity of the pathologic changes that take place in the cells of the nervous system. Following the destruction of the nerve-cells, secondary degeneration occurs. As the result of the pathologic process small or large areas of sclerotic tissue or cysts are formed. The size of these areas may vary from patches so small as to be hardly discernible to the naked eye, to destructive processes in which an entire lobe or hemisphere is involved. In those cases in which the pathogenic changes occur on the surface of the brain there is frequently an involvement of the meninges, which later on causes adhesion of the membranes to the cortex.

Bacteriology.—In most cases the organism causing the disease may be found in the foci of inflammation in the brain.

Symptoms.—The disease usually develops acutely. Children are affected more frequently than adults. For a day or two the patient may complain of headache, irritability, and a general feeling of illness. Either with or without these symptoms there may occur a sudden chill, followed by a rise of temperature to 100° to 104° F. The temperature remains persistently high, and the pulse may be rapid but regular. The respiration may be regular or of the Cheyne-Stokes type. A leukocytosis is generally present. Later, mental confusion and delirium develop. There may be stiffness of the neck, convulsions, or unconsciousness. The coma is rarely so deep that the pupillary reflexes, tendon reflexes, and the superficial reflexes are lost.

In some cases the delirium is marked, and may occur only as the stupor disappears. The patient may die during the first day or two, the coma gradually becoming deeper with the increasing temperature. In other cases the disease may run a more protracted course, the patient remaining more or less constantly unconscious, or the stupor may alternate with delirium, the temperature remaining high, and the pulse being rapid and irregular. Respiratory disturbances occur, and death intervenes within two or three weeks. In other cases there is a

gradual subsidence of the symptoms, the unconsciousness becoming less deep, the temperature decreasing, and the general constitutional symptoms disappearing. It is in these cases that, within a few days after the onset of the illness, symptoms appear pointing to interference with some portion of the cerebral substance.

If the brunt of the disease has fallen on the frontal lobe or its conduction pathways, a state of imbecility develops, and the individual, who before his illness was bright, may after recovery be mentally deficient.

If the precentral convolutions are involved, weakness or paralsyis occurs. If the occipital lobes are affected, visual disturbances occur; if the temporal lobes are involved, disturbances of hearing may occur; if the cerebellum is involved, there may be disturbances of equilibrium, ataxia, nystagmus, etc. In those cases in which the inflammatory involvement is limited to the crura and the adjacent structures, the local symptoms are confined to the eye muscles, and there occur ptosis, strabismus, opthalmoplegia, nystagmus, etc. In addition to these symptoms there may be ataxia, optic neuritis, tremor, disturbances of speech, staggering gait, etc. This type of case is described by Wernicke as polioencephalitis superior. In some cases the inflammatory condition may be limited to the lower pons and medulla; when this is the case the symptoms are those of a bulbar palsy of acute onset, and there occur disturbance of speech, swallowing, and respiration, frequently accompanied by spastic paralysis or weakness of all four extremities, or by disturbance of sensation in the parts below the lesion. This type of case is described under the title of polioencephalitis inferior.

Any combination of symptoms may occur, dependent upon the severity, the distribution, and the extent of the inflammatory condition.

Course and Prognosis.—Some cases begin with high temperature, marked constitutional symptoms, coma or delirium, paralysis or convulsions, etc. They may terminate fatally within one or two days. Other cases may likewise begin with marked constitutional disturbances, accompanied by delirium

or coma, convulsions or paralysis, which symptoms may continue for several weeks, a fatal termination occurring as the result of inanition or cardiac or respiratory disturbances.

Those cases presenting symptoms of a polioencephalitis inferior usually terminate fatally within a short time as the result of cardiac or respiratory disturbances. Cases that do not manifest such marked elevation of temperature at the onset, and in which the coma is not so deep, and in those cases in which convulsions are absent, usually after a short time the temperature subsides and the coma disappears. As time passes a gradual improvement sets in, until finally the prominent symptoms become very limited in extent. Unfortunately, epilepsy is a somewhat common sequel of this affection.

Diagnosis.—The diagnosis is made from the acute febrile onset, and from the constitutional symptoms, which are rapidly followed by manifestations pointing to local cerebral disease. The diagnosis of acute non-suppurative hemorrhagic encephalitis should be made only with great caution. This disease must be differentiated from the cerebral symptoms that occur especially in children at the onset of the various infectious diseases, such as influenza, typhoid fever, pneumonia, measles, etc., which may be toxic in origin, and from sinus thrombosis and acute serous meningitis.

Acute non-suppurative hemorrhagic encephalitis is distinguished from the early cerebral toxic states accompanying the infectious diseases by the fact that the focal cerebral symptoms usually develop early, are well marked, and remain well defined, whereas in the toxic or irritative conditions the focal symptoms are more diffuse, not so well marked, and variable. From sinus thrombosis this condition is differentiated by the absence of local edema and the subacute onset of symptoms, pointing to a localized cerebral edema and destruction, the examination revealing the possible source of a sinus involvement. From an acute serous meningitis differentiation is made by the fact that the temperature is less marked as compared with the symptoms, the irritative manifestations, such as cervical rigidity, hyperesthesia, delirium, and convulsions, being more pronounced in serous meningitis. In encephalitis the temperature is higher

and the symptoms are more marked, and are not so diffuse, but more constant. The former disease is more likely to occur in older individuals and in those in whom there is a history of alcoholism, or who are suffering from chronic nephritis, etc. The latter disease is more prone to occur in children and in those who are suffering with some acute febrile illness.

Treatment.—During the acute stage ice-caps should be applied to the head, and cold sponges or packs be ordered to reduce the temperature. Free purgation by the adminstration of calomel, followed by a saline or by castor oil, is indicated.

In well-nourished individuals bleeding may be advisable. The patient should be kept in a quiet, well-ventilated room, and if he is very restless, sedatives may be given. After the acute stage has passed the treatment of the subsequent symptoms is the same as that indicated for the relief of symptoms the result of cerebral circulatory disturbances.

INFANTILE CEREBRAL PARALYSIS

This condition manifests itself during the first few years of life, the symptoms being due to a more or less extensive destruction of the brain substance.

Etiology.—The brain may be affected during fetal life, but is more frequently involved during labor or within the first few years of life. Heredity plays but little part in this condition, although there may be a tendency to the transmission of a defective cerebral development. During fetal life there may be an actual deficiency in the development of the brain, or the brain may be injured as the result of trauma to the gravid uterus. Inflammatory affections may involve the fetal brain, and states of malnutrition, shock, injury, marked emotional disturbances, infectious diseases, chronic intoxications, etc., affecting the mother may interfere with the normal development of the fetal brain. During labor the infantile brain may be compressed by forceps, or prolonged labor may bring about circulatory disturbances and compression. Interference with the circulation by compression of the umbilical cord, rupture

of the cerebral vessels, etc., may bring about pathologic states of the infantile brain.

After birth and within the first few years of life the development of the infantile brain may be interfered with as the result of injury, inflammatory states secondary to infectious diseases, toxic conditions, malnutrition, circulatory disturbances, syphilis, diseased states of the ductless glands, convulsions, etc.

Pathology.—The pathologic changes vary much in distribution and in character, and are largely dependent upon the cause of the cerebral disease.

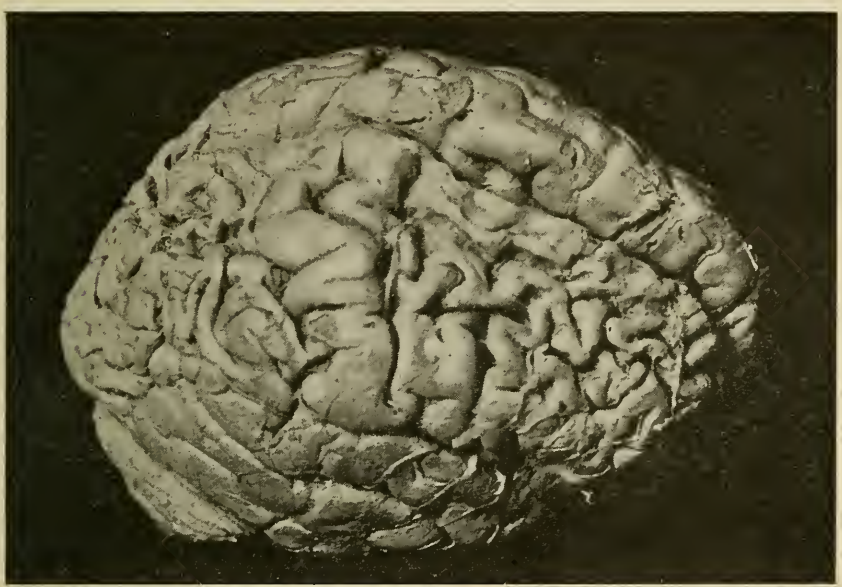


Fig. 142 —Brain from a case of infantile cerebral paralysis.

The causes that act during intra-uterine life most frequently bring about large cerebral defects, which result in lack of development of a portion of the brain. The undeveloped area may be occupied by a cyst, giving rise to a true porencephalitis. There may be a defective development of a portion of the brain, with a decrease in size of the convolutions and a lack of development of the cellular elements; in other cases there may be deficient development of the entire brain and of the cellular elements (Fig. 142).

As the result of injury, disease, or nutritional disturbances of the mother there may occur injury or disease of the fetal brain, which may give rise to localized or diffuse destructive conditions, followed by lack of development, cystic formations, or sclerotic areas. The infantile brain may be injured during labor, meningeal or subpial hemorrhages occurring, with subsequent compression or softening, followed by lack of development with the formation of scar tissue or the development of cysts. After birth, as the result of hemorrhage, occlusion of the cerebral vessels, inflammatory conditions, and the like, there occurs a deficiency in the development of the brain. This may be more or less extensive, depending upon the cause, and be accompanied by the formation of scar tissue, cysts, or the development of a hydrocephalic condition.

Symptoms.—If the causative condition occurred prenatal or during labor, symptoms pointing to defective functioning of the brain manifest themselves either shortly after birth or within the first few months of life. If the causative condition occurs postnatally, the symptoms pointing to cerebral involvement occur along with the evidence of a more or less acute cerebral involvement.

Owing to the fact that the pathologic lesion may be limited in extent there occur three types of cases: one group of cases is characterized by the prominence of motor symptoms; another group by the preëminence of mental defects; and a third group by the prominence of sensory defects. Cases also occur in which symptoms are present pointing to a more diffuse cerebral involvement, marked by the presence of motor, sensory, and mental manifestations, or any combination of the three. Epilepsy may occur with any of these groups.

In those cases developing during intra-uterine life there is noticed, shortly after birth, difficulty in nursing, and as time goes on the child presents either evidence of a lack of mental development or manifestations of sensory defects, such as blindness or deafness. In addition, there may be observed a stiffness and limitation of movement of one or both sides of the body, affecting the upper and lower extremities of one or of both sides. Those cases that develop during labor usually present

evidences, shortly after birth, of some acute cerebral circulatory disturbance. The infant may breathe with great difficulty, is cyanotic, does not cry, may display convulsive movements, which may be limited or general, and cannot be made to take the breast. It is drowsy, stupid, or unconscious, and may remain in this state for several days or longer, later on becoming very irritable and restless and crying more or less constantly. Subsequently symptoms will arise pointing to localized cerebral involvement.

Those cases developing during the first few years of life more frequently have an acute onset, with a temperature ranging from 100° to 104° F., delirium or coma, headache, vomiting, and sometimes convulsions. Symptoms may occur pointing to meningeal irritation. As the acute illness subsides evidences of more or less serious disturbance of the cerebral functions will be noticed. Finally, these cases resemble those that occur during labor or prior to birth.

The type in which the motor symptoms are the most prominent will manifest a paralysis or weakness that may be more or less extensive in its distribution. Some cases are hemiplegic, whereas others are diplegic. This occurs usually in the prenatal cases and in the newborn. The upper extremity is, as a rule, more seriously affected than the lower extremity, and as the child grows older it often learns to use the affected lower extremity or extremities. The face is generally involved, although the automatic movements are not affected. Many of these patients show speech disturbances and mental defects, and some are subject to convulsions (Fig. 143).

The paralysis is always of a spastic type. The tendon reflexes are increased, particularly those of the affected extremities. Usually there is noticed a tendency to muscle contracture in the affected part; this may be persistent and keep the limbs flexed in a certain position—the upper arm may be held close to the trunk; the elbow may be flexed; the wrist may be flexed or extended; and the fingers may be flexed at all their joints and held clenched, or may be extended at the interphalangeal joints. The leg is usually slightly flexed at the knee-joint, and the foot is extended, with either eversion or inversion.

In other cases, instead of the contracture being persistent, voluntary movements may lead to overaction and tonic spasms, constant muscle tension being present and increasing with voluntary movement. In some cases muscle stiffness with overaction upon voluntary movements is the only evidence of the cerebral involvement. In a few cases associated movements

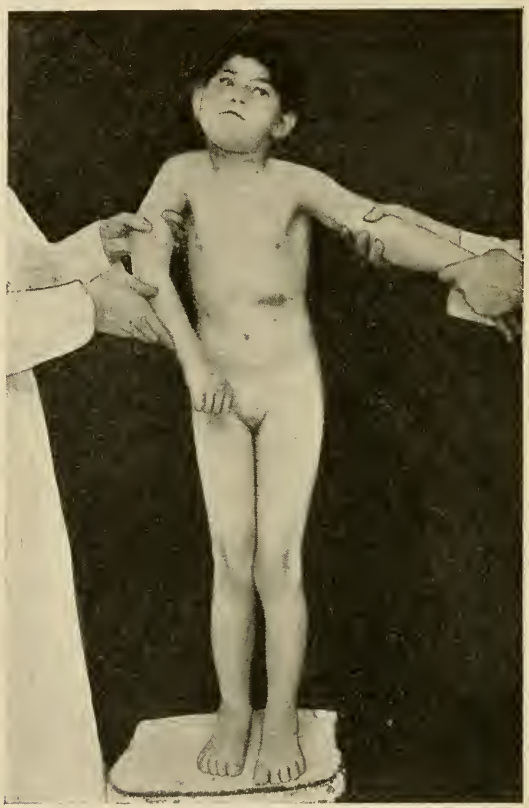


Fig. 143.—Infantile cerebral paralysis.

occur: thus the voluntary movements of the unaffected extremity are accompanied by more or less similar involuntary movements on the affected side.

The weakened or paralyzed extremities are also subject to peculiar, worm-like contractions which affect the fingers and hand more frequently than other affected portions; these are called athetoid movements. In some cases peculiar purposeless

involuntary contractions of a choreiform nature occur, involving the affected extremities.

The abnormal movements sometimes involve the face, but the upper extremities are always more markedly affected. These abnormal movements are usually intensified upon voluntary attempts to use the affected portion of the body.

In one form of infantile paralysis known as *Little's disease* spastic paralysis or weakness affects principally the lower extremities and to a much less extent the upper extremities. There is usually little or no mental enfeeblement, and no tendency to athetoid movements or epileptic attacks exists. This condition usually occurs in a child born after difficult labor, or in one who is born prematurely and is abnormally small. As the child develops a spastic condition involving principally the lower extremities is observed, and when efforts are made to walk there is noticed a strong adduction of both legs. As the steps are taken the legs become crossed, giving rise to a condition known as "scissors gait."

As the individual grows older it is seen that the affected limbs do not develop to the same extent as the unaffected limbs do. This lack of development affects the part *in toto*. There are no qualitative changes in the electric reaction, and sensory disturbances are rarely present.

Still another group of cases are seen in which mental enfeeblement is the most prominent symptom. In these there is noticed, either after an acute illness or dating from birth, a lack or absence of mental development. All grades of mental deficiency, from slight peculiarities of character and mild weakness of the intellect to idiocy, are observed. In the third group of cases the prominent symptom is blindness or deafness. In some cases the existence of homonymous hemianopsia is discovered on examination. In those cases in which deafness has occurred before the individual has learned to talk speech is never acquired and thus deaf-mutism exists.

Many of the cases that show motor symptoms also present evidence of some mental deficiency, which may range from the very mildest grades to absolute idiocy (Fig. 144). Convulsive seizures are of frequent occurrence in those cases in which

mental or sensorial defects are the prominent symptoms. Mental enfeeblement frequently occurs along with the sensorial defects.

Amaurotic family idiocy, according to Sachs, is usually present in several members of a family. The children in such

Fig. 144.—Infantile cerebral paralysis, showing motor and intellectual defect (case of Dr. G. E. Bennett).

a family present at birth a healthy physical and mental development, which continues for several months, when a change sets in, and they become weak and stupid. Gradually blindness develops, due to optic atrophy (Fig. 145).

Prognosis.—Complete recovery is not to be expected. The

underlying pathologic condition is not of a progressive nature, except in the type of disease known as amaurotic family idiocy. As a consequence, there is no progression of symptoms, except in amaurotic cases. The sensory defects usually do not improve,

Fig. 145.—Amaurotic family idiocy.

although there may be slight improvement in the intellectual and the motor defects. In Little's disease considerable improvement may take place.

The convulsive seizures which occur may be held in check

and their severity decreased by proper medication, but their occurrence cannot be absolutely prevented. These individuals seem particularly susceptible to acute illness, and the greater number of cases die before reaching their thirtieth year.

Diagnosis.—The diagnosis of infantile cerebral palsy usually presents no difficulty. It is based on the history of the case, on the presence of weakness, paralysis, or stiffness of a hemiplegic or a diplegic character, in conjunction with mental enfeeblement, more or less marked, and at times sensory defects, abnormal movements, and epilepsy. In these cases, even when there is a decrease in the size of the affected extremities, the electric reactions are qualitatively normal and the tendon reflexes are increased.

The diagnosis of Little's disease is made from the presence of a spastic weakness or paralysis of all four extremities, the lower being affected to a much greater extent than the upper; the absence of mental defects and of epilepsy; and the fact that the affection manifests itself within the first few months of life. The diagnosis of amaurotic family idiocy is made from the presence of a progressive spastic weakness or paralysis, hemiplegic or diplegic in distribution, accompanied by optic atrophy, with a peculiar fundal change (Fig. 146). The fact that the affection occurs usually in several members of a family during the first few months of life is also of diagnostic value.

Treatment.—The treatment of this condition is largely prophylactic in nature; the pregnant mother should be properly nourished and cared for during the period of pregnancy, should be spared all unnecessary emotional disturbances, and guarded against trauma, etc. During labor the head of the child should be properly protected, and the delivery should be effected under proper medical supervision, so as to prevent undue pressure of the head or a too prolonged labor. In other cases, after prolonged or difficult labor, when the presence of a subdural hemorrhage is suspected, the patient should be carefully watched, and if convulsions or stupor occur, surgical intervention is to be seriously considered.

The treatment of the acute cerebral conditions occurring in early childhood, such as meningitis, encephalitis, hemorrhage,

thrombosis, etc., is the same as that which has been described in the previous chapters.

Later the treatment should be directed toward preventing the development of contractures; this is best accomplished by massage, passive movements, and the faradic current applied to the weaker muscles. If, in spite of these measures, contractures still tend to occur, they should be overcome by the application of splints or of different forms of orthopedic apparatus. In cases in which the abnormalities of movement seriously

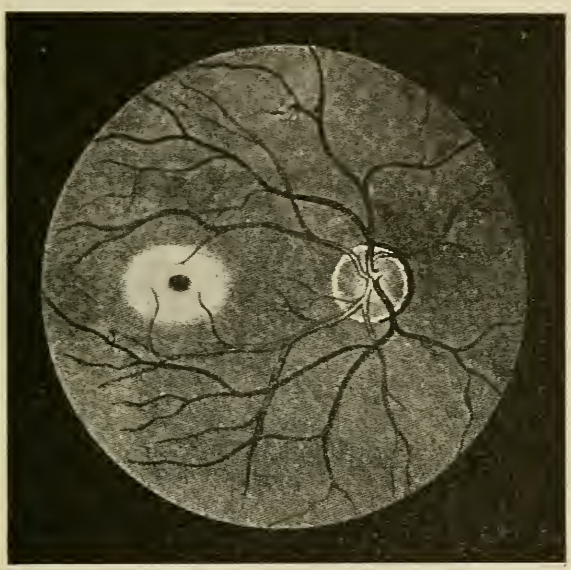


Fig. 146.—The fundus in amaurotic family idiocy.

interfere with the comfort of the patient, the intradural section of several posterior nerve-roots by Foerster's method may give relief. In some cases the mental defects can be improved by education; in other cases nothing can be accomplished. The blindness and deafness are not amenable to treatment, although some deaf patients may be helped by those accustomed to teaching the deaf and dumb.

Epilepsy can be controlled to a certain extent by the use of bromids or other sedatives.

The patient should be kept clean, and protected from ex-

posure. The diet should be light but nutritious, and proper supervision of the bowels and bladder should be maintained. Every effort should be made to see that the patient is cleanly in his habits. If there is reason to believe that an increased intracranial pressure exists, especially if choked disc is present, cerebral decompression is indicated. In many cases in which this was performed the patient has been benefited. When the examination reveals the presence of a local cortical irritative lesion, operation is justifiable. Each case should be carefully considered as to the advisability of performing a cranial operation, for in the vast majority of cases this procedure is absolutely useless. In most cases the best results are had from careful nursing, proper attempts at education, relief of the epilepsy, the placing of the patient amid agreeable, cheerful, protected surroundings, relief of the abnormal movements, and the prevention, so far as possible, of unfavorable positions of the limbs by means of massage, electricity, mechanical appliances, or operative procedures.

HYDROCEPHALUS

Etiology.—This condition may be congenital or acquired. Chronic intoxication, syphilis, acute infectious diseases affecting the mother during pregnancy, trauma, and heredity seem to be the most common predisposing conditions in the development of a congenital hydrocephalus. In the acquired form the condition may have been slight at birth, and remained latent for some time, developing later as the result of injury, exposure, etc. An acquired hydrocephalus may be secondary to tumors that exert pressure upon the veins of Galen or upon the aqueduct of Sylvius, or it may be secondary to inflammatory diseases of the meninges that close the foramen of Magendie, and thus prevent the flow of cerebrospinal fluid into the subdural space. In other cases there is an increase of the cerebrospinal fluid in wasting diseases, in nephritis, and in atrophy of the convolutions of the brain in old age. The condition may also be secondary to a serous meningitis of the ventricles, in which case it may run an acute or a chronic course. This variety is believed

to be due to injury, exhaustion, chronic toxemia, pneumonia, typhoid fever, tuberculosis, and, less frequently, other infectious processes.

Pathology.—This condition is marked by the presence of an excessive quantity of fluid either in the cerebral ventricles, giving rise to an *internal hydrocephalus*, or between the brain and the dura, giving rise to an *external hydrocephalus*. The fluid is generally clear and colorless, and contains but little albumin and a small amount of lime salts. In some cases the hydrocephalus may be limited to a single ventricle; as a rule, the fourth ventricle is least affected. The development of the

Fig. 147.—Hydrocephalus.

brain is almost always retarded, and in some cases the walls of the hemisphere may be merely a layer several millimeters in thickness, the convolutions and sulci being completely obliterated (Fig. 147).

In the congenital cases, and in those developing before ossification of the sutures of the cranial bones has taken place, the skull is increased in size and the cranial bones are reduced in thickness. The head becomes irregularly globular in shape, with prominent frontal and parietal protuberances. The choroid plexuses are frequently enlarged. In some cases an ependymitis of the ventricles is found, with thickening of the ventricular lining, closing the foramen between the ventricles; at times

there is a closure of the foramen of Magendie. Defective development of the cerebellum and of the brain-stem is occasionally observed. Those cases that occur after ossification of the cranial sutures do not show any increase in the size of the head, and the amount of fluid present is smaller. In these cases the pressure is exerted more directly upon the brain substance, with subsequent distruction of the brain tissue.

Symptoms.—If the hydrocephalic enlargement of the head is well marked before birth, labor is rendered difficult, and is sometimes impossible without a mutilating operation. In those cases in which the condition is not pronounced at birth it usually appears during the first six months or year. When the condition develops after an acute or a chronic illness, before ossification of the cranial sutures has taken place, there occurs a more or less rapid enlargement of the head. The internal pressure is manifested by the bulging of the fontanels, which increase in size. As the condition progresses there is a marked disproportion between the size of the skull and the face; the frontal and parietal protuberances project over the face and ears; the orbits are frequently directly downward instead of forward, and the veins of the scalp are prominent.

In time there is noticed a defective development of the intellect. Optic atrophy, nystagmus, and strabismus are very common. These children are usually inactive, and may be restless and irritable. In other cases there is a marked tendency to the development of stuporous states. Many of these children cannot learn to speak, or if they do, speech is acquired late and is often defective. Vomiting is frequently present, or may be induced by attempts at movement. Such children learn to walk late, if at all. Frequently spastic weakness or paralysis is present on one or both sides, more often in the legs, and convulsive seizures are not uncommon.

When an internal hydrocephalic condition occurs after closure of the sutures and fontanels, the symptoms are those pointing to increased intracranial pressure, *e. g.*, pain, headache, vomiting, vertigo, insomnia, restlessness, stuporous states alternating with delirium, spastic paralysis, choked disc, strabismus, and sometimes nystagmus and pupillary disturbances. A

slight exophthalmos is usually present, and rarely there is a spontaneous escape of cerebrospinal fluid through the nose.

Course and Prognosis.—The duration of the congenital cases varies considerably; most cases run a rapidly fatal course, the patient dying within the first few months of life. Other cases in which the hydrocephalus is not so well marked last a longer time, and the patients are capable of a fair amount of mental development. Very mild grades of hydrocephalus also occur in which spontaneous arrest of the condition may take place. These individuals are capable of normal mental development and may attain old age. From my experience it would appear that those cases which are most marked and which develop soon after birth lead to a rapidly fatal termination. The course and prognosis of an acquired hydrocephalus are dependent principally upon the cause; as a rule, the outlook is rather grave, although in some cases spontaneous arrest of the condition takes place.

Diagnosis.—Those cases accompanied by hydrocephalic enlargement usually present no difficulty in diagnosis. The condition may be differentiated from rickets by the globular shape of the head in hydrocephalus in contradistinction to the rectangular shape of the head in rickets; the absence of other signs of rickets in hydrocephalus, and their appearance in the long bones and costal extremities in rickets. In later life hydrocephalus must be differentiated from hyperostosis cranii; in the latter condition the increase in the size of the head is due to thickening of the cranial bones, which can be detected by *x*-ray examination. In this disease the bones of the face and spine are also usually thickened. From acromegaly hydrocephalus is differentiated by the fact that in the former condition there is an associated enlargement of the hands and feet, a marked enlargement of the lower jaw, and a thickening of the frontal bones. The diagnosis of a hydrocephalic condition after ossification of the cranial bones has taken place is a matter of great difficulty in those cases in which no enlargement has taken place. In these cases the diagnosis of the causative condition is usually made, and the presence of the hydrocephalus is inferred.

Treatment.—In those cases in which syphilis is suspected as being a factor in the causation of the condition mercurial inunctions and potassium iodid internally may be of value. Cold applications should be made to the head and the bowels be kept well open. The amount of liquids ingested should be limited. In those cases in which there is restlessness, pain, or convulsions, relief may be had by the administration of sedatives. Treatment of the underlying condition is of the utmost importance. If the hydrocephalus is the result of a meningitis, with closure of the foramen of Magendie, benefit may result from a suboccipital operation and the breaking up of meningeal adhesions. In those cases due to pressure by a tumor operation for removal of the growth must be considered. The surgical treatment, consisting of repeated ventricular puncture and ventricular drainage, strapping of the skull, etc., has been very unsatisfactory.

ABSCESS OF THE BRAIN

The formation of a brain abscess is always secondary to a purulent encephalitis produced by microörganisms. Trauma to the head is one of the common causes of cerebral abscesses—usually the injury produces a fracture, with subsequent suppuration of the wound and secondary involvement of the brain. Infected wounds of the scalp may also be a cause of cerebral abscess. An abscess may form on the brain shortly after an injury, or an interval of some time may elapse before the cerebral symptoms manifest themselves, the health remaining comparatively good in the meantime.

Another very common cause of brain abscess is chronic suppurative middle-ear disease. In these cases there is usually a history of repeated attacks of otitis media, or of the presence, at some previous time, of a purulent discharge from the ear. In these cases erosion and infection of the bones of the tympanic cavity or of the mastoid cells occur: in the former case the temporal lobes are infected, and in the latter case the infection invades the cerebellum. The abscess may communicate by means of a fistula, through the thickened and inflamed dura,

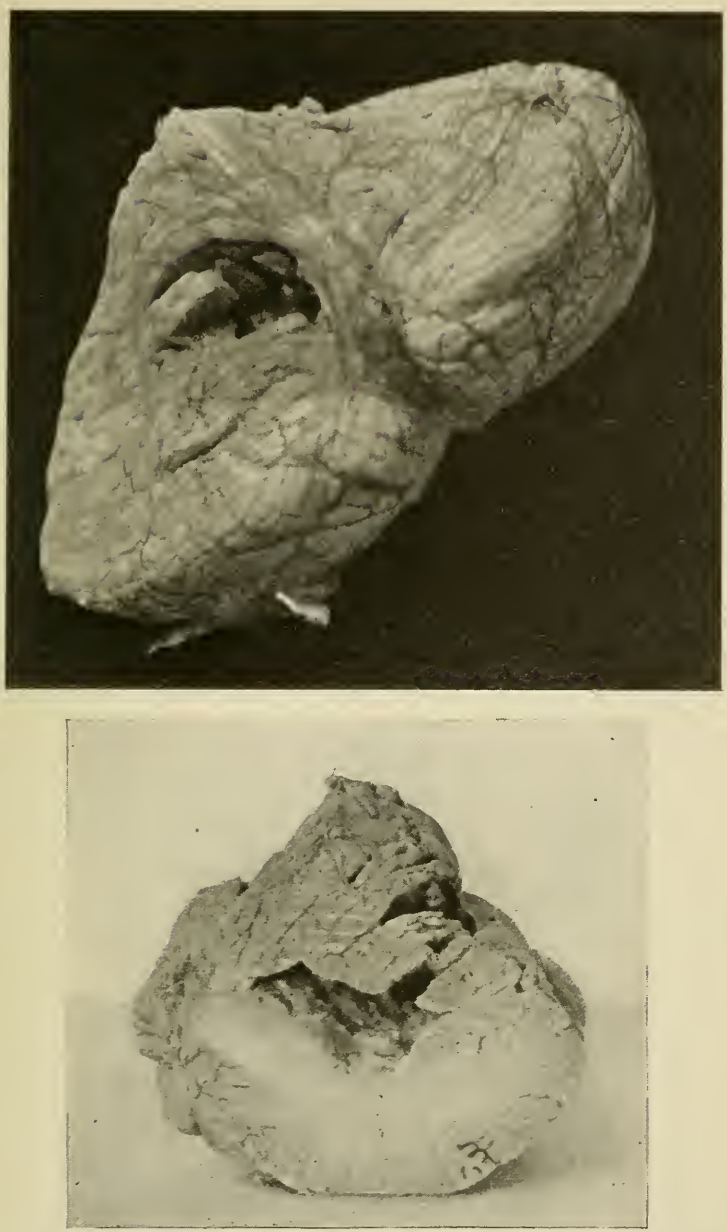


Fig. 148.—Abscess of the cerebellum (case of Dr. Charles Bagley).

with the bone lesion, but in most cases there is a layer of brain tissue between the abscess and the bone lesion, the infecting

organism invading the brain by means of the perivascular lymphatic space. Rarely brain abscesses are secondary to suppurative processes affecting other cranial bones, pyogenic infection of the nasal cavity or orbit, erysipelas, etc. In some few cases brain abscesses occur in general pyemic conditions secondary to ulcerative endocarditis, puerperal infection, suppuration in the bones or joints, gangrene in the lungs, etc. In this latter group of cases multiple abscesses, as a rule, develop. The abscess may also occur in connection with one of the infectious diseases, such as typhoid, influenza, epidemic cerebrospinal meningitis, measles, etc.

Cerebral abscesses occur more frequently in males than in females, and are most common between the tenth and the thirtieth year.

Pathology.—In cases due to trauma the abscess is usually situated in the subjacent brain tissue. When secondary to otitis media, the abscess is located on the same side of the brain as the ear that is affected, and is generally either in the temporal lobe or in the cerebellum (Fig. 148). In rare cases a localized encapsulated abscess involving the dura and the cortex may occur; when such abscess forms, it is found either in the middle or in the posterior fossa of the skull. Streptococci, staphylococci, pneumococci, the influenza bacillus, typhoid and tubercle bacilli, etc., have all been found in cerebral abscesses.

Cerebral abscesses may be small or large, encapsulated or irregular in outline, and single or multiple. They may be of rapid or of slow growth. In the earlier stage there is a localized purulent encephalitis, followed by a gradual breaking down of brain tissue, with the formation of pus within an irregular cavity. This cavity has no distinct walls, but is surrounded by brain tissue that is more or less affected, and that contains hemorrhagic areas. In other cases there is a more or less definite wall formation, and even in these cases there may be an involvement of the brain tissue beyond this wall, with subsequent extension of the abscess. The majority of abscesses that have existed for any considerable length of time are surrounded by a definite capsule. This capsular wall begins to form as early as the second week, and if the abscess persists for

some time the wall may become thick, resistant, and even calcify. In many cases, owing to the fact that the brain tissue surrounding the abscess is also infected, there is a breaking down of the walls, with an extension of the abscess; thus the abscess may rupture into the ventricles or on to the surface of the brain.

The pus of a brain abscess is usually green or yellow in color, often has an extremely disagreeable odor, and sometimes contains disintegrated brain tissue.

Symptoms.—The symptoms of the early stages of brain abscesses are usually masked by those of the causative condition, and hence are often overlooked. A careful consideration of the history of brain abscesses will permit one to distinguish three stages, which sometimes merge rapidly, and at other times very slowly, into each other. In the first or stage of invasion, there occur the symptoms of the underlying disease, together with evidence pointing to involvement of the brain tissue, such as localized or diffuse headache, restlessness, stupor or delirium, irritability, indifference, rarely choked disc, etc. In this stage the temperature may be high, subnormal, or irregular; chills and profuse sweats may occur, and a leukocytosis is usually present. This stage, which may continue for from a few days to several weeks, may be followed by the second or third stage.

If the second stage follows, there is a remission of symptoms, and the patient enters the latent stage, in which the headache is less severe or disappears, the temperature remaining subnormal or showing an occasional rise. In this stage intense headaches may arise without apparent cause, and there may be a change in the mental characteristics of the patient—he may become irritable or indifferent, or exhibit a gradually increasing mental dulness and slowness of thought.

The patient may remain in this stage for a period varying from a very short time to many years, when, more or less suddenly, the symptoms of the third stage may be ushered in, or the second stage may gradually pass into the third stage.

In the third stage the headache becomes more persistent and severe, and vomiting and vertigo may occur. The tem-

perature remains subnormal; the pulse becomes slow, the beats ranging from 50 to 70 a minute. Convulsive attacks may occur, together with paralysis of the cranial nerves, optic neuritis, etc.

In some cases the stage of invasion passes more or less rapidly into the terminal stage. In addition to the symptoms of the causative condition, the patient becomes stuporous or delirious, and headache and vomiting occur. The temperature may be very high, the pulse irregular, and leukocytosis, chills, convulsive seizures, etc., may supervene. The patient becomes emaciated and there is great prostration; symptoms of meningeal involvement may set in, and death terminates the condition.

There is another group of cases in which, after the stage of invasion, there occurs a period during which the patients are apparently without symptoms for a considerable length of time, varying from months to many years. These patients may gradually develop symptoms suggestive of cerebral tumor, but which are accompanied by subnormal or unexplainable rises of temperature, leukocytosis, and at times chills or general septic manifestations. Other patients in the latent stage may suddenly, without premonitory symptoms, develop an acute attack resembling a stroke of apoplexy; this may be ushered in by convulsive seizures, and be followed by rise in temperature, leukocytosis, chills, delirium or stupor, nystagmus, pupillary inequalities, etc., death occurring within a short time.

Diagnosis.—In formulating a diagnosis of cerebral abscess the history of a focus of infection or the appearance of one of the commoner preëxisting causes of brain abscess is of the greatest importance. When there is no history or evidence of a probable cause for the condition only a tentative diagnosis can be made. If, shortly after an injury to the skull, there be added to the local manifestations symptoms pointing to a local cerebral disturbance in an area corresponding closely to the traumatic lesion,—symptoms accompanied by fever, chills, leukocytosis, etc.,—the diagnosis of a cerebral abscess or of a meningitis may be made. It is a matter of great difficulty in these cases to distinguish between these two conditions: In meningitis the irritative symptoms are more marked; the

temperature is apt to be higher; the pulse is rapid and irregular; there is a tendency for the disease to spread rapidly; cervical rigidity and cranial nerve palsies occur. In cerebral abscess the symptoms are more apt to remain localized; temperature is less likely to be high; the pulse becomes slower, and stupor or coma develops; and if there is much increased intracranial pressure, choked disc occurs.

In those cases in which cerebral symptoms occur some time after an injury to the head differentiation must be made between cerebral abscess, brain tumor, and traumatic neurosis. Traumatic neurosis is characterized by the absence of fever, of slow pulse, and of focal symptoms, and by the presence of sensory and vasomotor symptoms, and subjective vertigo. There is also marked variation of symptoms, and usually the evidence of a neuropathic disposition. In brain tumor the development of the symptoms is more apt to be insidious; choked disc is frequently present early; fever and leukocytosis are generally absent, and the focal symptoms, when present, show a gradual but regular tendency toward progression. In cerebral abscesses manifesting themselves some time after an injury the onset is, as a rule, more sudden, and is accompanied by temperature, chills, and leukocytosis. Remissions occur more or less frequently, and the progress of the condition is irregular.

If a cerebral abscess is secondary to a chronic otitis media, it must be differentiated from thrombosis of the lateral sinus and meningitis. From thrombosis of the lateral sinus it is differentiated by the presence, in thrombosis, of a swelling, tenderness, and edema of the mastoid process; tenderness and swelling and sometimes a cord-like appearance of the jugular vein on the side of the neck. The temperature is generally irregular, ranging from the subnormal to 104° F. or over; chills are frequent, and the pulse is rapid and irregular. In meningitis the onset is usually more rapid; the irritative symptoms, such as hyperesthesia, cervical rigidity, convulsions, and delirium, are generally present; the temperature varies, but rarely goes below normal; the pulse is apt to be rapid, irregular, and intermittent; paralysis of the cranial nerves and optic neuritis occur early; leukocytosis is marked; examination of the spinal fluid reveals

the presence of microörganisms and leukocytes. In abscess the onset is usually less sudden, the irritative symptoms are generally absent, the temperature is frequently subnormal, the pulse is slow, and choked disc occurs later; local cerebral symptoms which are very slowly progressive are prominent, and the spinal fluid does not show the presence of leukocytes or of microorganisms. Some cases of otitis media are accompanied by marked cerebral symptoms, without the development of brain involvement. These cases usually promptly clear up when proper treatment is directed toward the ear condition. The leukocyte count rarely goes above 12,000, and optic neuritis and symptoms pointing to local cerebral involvement do not occur.

The **diagnosis** of a cerebral abscess occurring during the course of the acute infectious diseases, septic conditions, in purulent diseases of the lungs, etc., is always made with difficulty. The presence of cerebral symptoms, which may be of acute onset, the occurrence of remissions, together with localized signs and evidence of increased intracranial pressure, should always arouse a strong suspicion of the presence of a cerebral abscess.

The diagnosis of the location of a cerebral abscess is always a matter of great importance, and should be made from the presence of the localizing symptoms. Cerebral abscesses following injury to the skull usually occur in the portion of the brain situated adjacent to the traumatic lesion. Abscesses secondary to middle-ear disease commonly involve the temporosphenoidal lobe or the cerebellum, and one cannot always determine with certainty the location of such an abscess. When one ear only is affected, the abscess is always on the side of the affected ear. Examination of the ear to discover the site of the carious process is of great importance, as those abscesses that form subsequent to necrosis of the roof of the middle ear generally develop in the temporal lobe, whereas those which occur secondary to a necrosis of the posterior wall or of the mastoid cells usually involve the cerebellum.

The presence of cerebellar ataxia, stiffness of the neck, nystagmus, choked disc, or symptoms pointing toward involvement of the pons and medulla indicate cerebellar involve-

ment. Symptoms consisting of disturbance of hearing, hemianopsia, and sensory disorders point toward temporal lobe involvement. Suppurative processes secondary to septic conditions are usually multiple, and may give rise to no localizing signs.

Prognosis.—The outlook is grave, death occurring from the development of a cerebral edema or from the bursting of the abscess into the meninges or ventricles, and setting up a purulent meningitis. The probability of recovery of an individual who has a cerebral abscess is dependent mainly on the possibility of successfully evacuating the contents of such an abscess cavity, and upon the formation or the presence of an encapsulating membrane that will preclude extension of the infection to some adjacent cerebral tissue. A certain number of cases, even if properly opened and treated, will succumb as the result of the slow, intermittent extension of infection to the adjacent cerebral tissue and a failure of the abscess cavity to be completely obliterated.

Treatment.—The treatment of this condition is purely surgical. The cranial cavity should be opened over the suspected location of the abscess, the strictest aseptic precautions being observed. If time permits, it is advisable, by irritation of the meninges, to cause a closing off of the rest of the cerebral cavity by the formation of adhesions between the membranes at the operative site; then, in about twenty-four or thirty-six hours after this has occurred, the abscess cavity should be freely opened, and measures instituted to allow free drainage. This is most important, as unless the abscess cavity granulates or fills up from the bottom, a recurrence will take place after the drainage canal has closed.

CEREBRAL TUMORS

The brain, like other portions of the body, is at times the seat of new-growths. Under this head will be described all new formations taking place within the skull, and encroaching upon the contents of the cerebral cavity.

Etiology.—Tumors of the brain may occur at any age, but the greater number of cases occur during childhood and in young adult life. They are probably somewhat more frequent in males than in females. In some cases injury may be assigned as the exciting cause, but this is so in but very few cases. Some forms of cerebral tumor are secondary to tumors or disease in some other portion of the body. This is practically always the case in tubercular growths, in carcinomata, parasitic cysts, etc. Cerebral tumors may also occur as the result of direct extension of tumors of the cranial bones, pharynx, orbit, etc.

There is no satisfactory explanation as to the origin of many forms of tumors, such as glioma, fibroma, sarcoma, etc.

Pathology.—Tumors may be single or multiple. Certain forms of tumor, such as sarcoma and glioma, and some of the rarer varieties, are usually single. Other forms, such as tuberculomata, carcinomata, syphilomata, etc., are sometimes multiple. The tumor may involve almost any portion of the brain substance, the cortex or the substance immediately beneath the cortex being the point of origin in the greatest number of cases. The brain-stem and the cerebellum are involved with about an equal degree of frequency, but less often do tumors involve the basilar ganglia and the surrounding structures. Of the commoner forms of brain tumors may be mentioned tubercle, sarcoma, glioma, gliosarcoma, and gumma; less frequently carcinoma occurs, but cysts are present in a large proportion of cases. Among the rarer forms of tumor are fibroma, angioma, lipoma, psammoma, osteoma, neurofibroma, etc.

Tuberculomata occur most frequently in childhood, and are rarely found after the age of thirty. They may be single, but in a large proportion of cases more than one tumor is present. These tumors occur most frequently in connection with the meninges and large vessels, and consequently are generally found on the surface of the brain. They involve by preference the pons, the cerebellum, and the cortex of the hemispheres. They do not destroy the cerebral tissue, but compress and displace it, and are frequently surrounded by an area of tubercular infiltration. The tumor may vary in size from a small collection

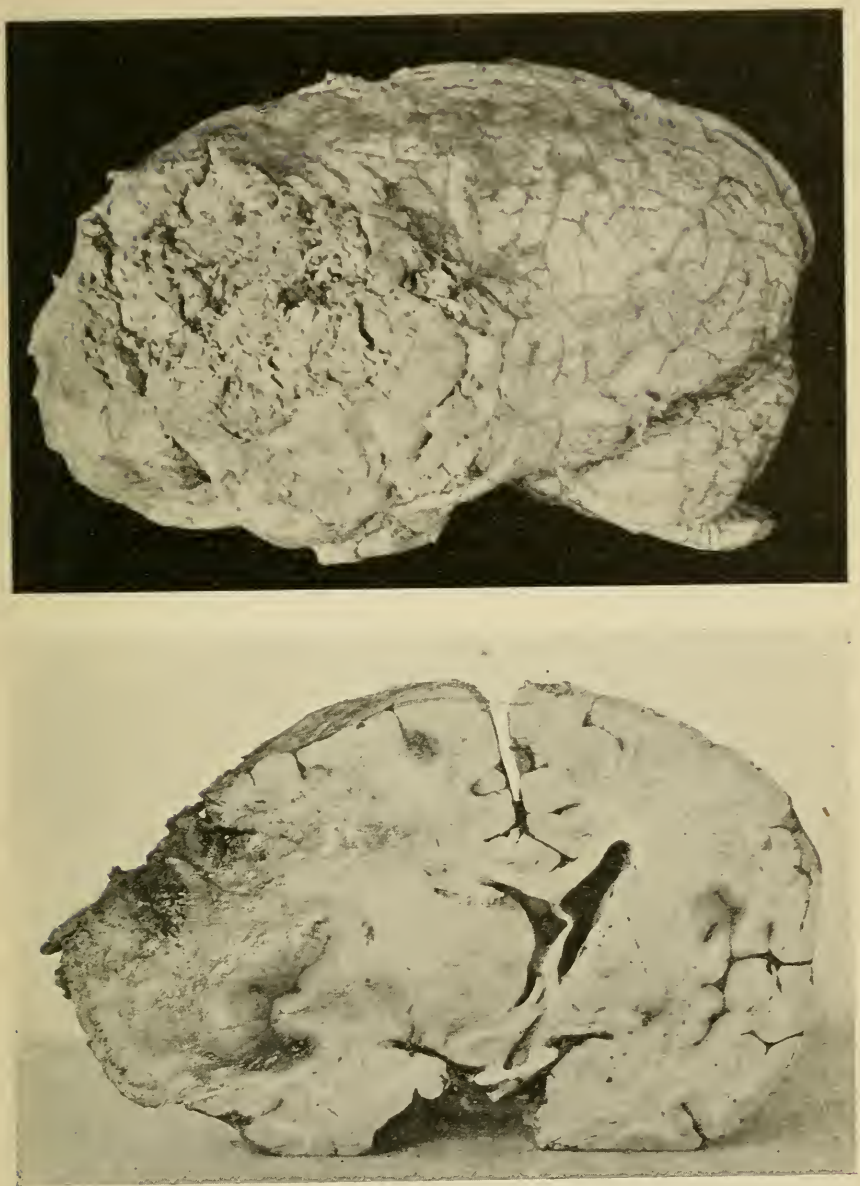


Fig. 149.—Cerebral tumor. Frontotemporal lobe (case of Dr. Charles Bagley).

of miliary tubercles, forming a small mass, to a large tumor which is hard and which may be definitely encapsulated.

Sarcoma usually arises from the meninges, the periosteum of the cranial bones, etc. As a rule, it is single and encapsulated, grows rapidly, and gives rise to well-marked symptoms. These tumors occur more frequently in the cortex of the hemispheres and cerebellum than in other localities. In some cases a sarcoma is not definitely circumscribed. These tumors do not usually infiltrate the brain substance, but merely displace and compress it, and when they are so situated as to be accessible to surgical intervention, can generally be successfully removed.

Glioma.—Gliomatous tumors are third in frequency of occurrence of all brain tumors. They vary in size from that of a small nut to almost the size of an entire hemisphere. These tumors are usually primary in type, and are peculiar to nervous tissue. They are not sharply defined from the surrounding brain tissue, but extend by infiltration, and cannot be removed without completely destroying the tissue which is involved (Fig. 149). These tumors are usually soft, having about the same consistency as the brain substance. There is a tendency for these tumors to undergo degeneration, so that cavities filled with either a yellowish or reddish fluid are frequently found in them. Some of these tumors are of a yellow, white, or gray color, and at times differ very little in color from the normal brain tissue. Gliomata are made up mainly of glia-cells and an intercellular fibrillar network. These tumors are usually very vascular, and hemorrhage into the growths is of common occurrence. Glioma may involve any portion of the brain, but occurs somewhat more frequently in the white substance.

Gliosarcoma.—This form of growth occurs less frequently than either glioma or sarcoma, the tumor partaking of the nature of both of these varieties. It infiltrates and destroys the brain tissue, and is rarely encapsulated.

Carcinoma.—This tumor is usually secondary to carcinoma in some other portion of the body, or occurs as a direct extension of carcinoma of the orbit or cranial or facial bones. It generally occurs in adults. Carcinoma is a soft, vascular tumor, of irregular form, and is not encapsulated. It is frequently multiple, and may involve any portion of the brain.

Syphilitic Tumors.—These tumors almost always arise from

the meninges, and show a tendency to spread flatly on the surface of the portion of the brain that is involved. They occur very rarely in childhood, but are somewhat common in adult life. They are multiple, vary in size, and rarely become larger than a hazelnut. There is no tendency on the part of these tumors to infiltrate the brain tissue, but they may be surrounded by an area of softened cerebral substance. These growths may develop within the first few years or many years after a syphilitic infection. They occur either as soft, gelatinous masses, or take the form of hard and more circumscribed tumors. They occur most frequently at the base of the brain.

Cysts of the brain arise either in connection with tumors, as the result of degeneration of the tumor mass, or following hemorrhage and softening. In some few cases the cysts are the result of anomalies in development of the brain. The contents of the cysts are sometimes watery or serous, and at other times they are viscid or colloid. In very rare cases dermoid cysts develop in the brain. Cysts are usually of slow growth, and produce symptoms by causing compression of the brain tissue. Only rarely do they destroy or infiltrate the cerebral tissue.

Psammoma occurs almost exclusively in the meninges and in the pineal gland. It is a new-growth containing abundant, chalk-like concretions, and develops very slowly. Other forms of tumor, such as fibroma, myxoma, lipoma, chloroma, etc., are of great rarity. As the result of the presence of a new-growth within the cranial cavity an increased intracranial pressure is produced. This increased pressure depends upon the rapidity of the tumor's growth and upon its location. Tumors under the tentorium usually produce a rapid increase of intracranial pressure, and as a result the membranes become tense and there may be a thinning of the bone. Tumors may be so situated as to produce obstruction to the return circulation from the ventricles, and thus bring about a hydrocephalic condition. There is usually a disturbance of the cerebral circulation, more or less cerebral edema, and, in the immediate neighborhood of the tumor, some destruction of brain tissue.

Tubercular and syphilitic tumors are usually accompanied by a more or less diffuse meningitis. In a few cases symptoms

resembling tumor are due to aneurysm of the large vessels, those at the base of the brain being the vessels most frequently affected.

Symptoms.—Symptoms of tumor of the brain are in many cases more or less definite, although cases do occur in which large tumors have existed without manifesting symptoms pointing to the condition. The symptoms of a brain tumor are dependent, first, upon the presence of a growth; second, upon the situation of the growth, third, upon the rapidity of growth of the tumor. As the result of the presence of a cerebral tumor two sets of symptoms arise—these are the general and the focal. The general symptoms are dependent upon the physical presence of a tumor, upon increased intracranial pressure, and upon the rapidity of its growth as well as upon its character. The focal symptoms are dependent upon the destruction or irritation of a limited cerebral area. In some cases topical symptoms exist.

General Symptoms.—Headache is one of the most constant and prominent symptoms of a brain tumor. It may be absent or only very mild in the early stages, but as the tumor increases in size or interferes with the ventricular circulation the headache becomes persistent and reaches a degree of severity rarely seen in other diseases. It is usually increased by physical effort, excitement, mental strain, coughing, etc. It is generally diffuse, but in some cases may be more intense in a localized area, such as the frontal or the occipital region. The headache is often associated with a diffuse tenderness over the entire head on percussion.

Stupor.—This is commonly absent during the initial stage, but cases occur in which, even from the onset, there is a marked tendency toward somnolence on the part of the patient. Thus he goes to sleep more or less constantly, sometimes even while eating. As a rule the patient can be aroused, will answer a question, and then immediately fall asleep again. Later in the disease the stupor becomes deeper and the patient is more difficult to arouse. Mental disturbances occur with a considerable degree of frequency; some individuals become irritable and impatient, whereas in others the disposition under-

goes a change and the patient loses interest and becomes indifferent. Mental dulness and apathy are frequently very noticeable. In some patients peculiar attempts to be facetious are observed.

General Convulsions.—These occur in one-half of the cases at some period of the disease. The convulsive seizures resemble epileptic attacks, and if they occur, as they sometimes do, before other symptoms appear, it is impossible to distinguish them from true epileptic fits. The convulsive seizures are usually preceded by some of the other general symptoms of a cerebral tumor. They vary in degree, in some individuals resembling attacks of petit mal, whereas in others there occurs a localized twitching in some parts of the body, or disturbance of consciousness is attended by automatic or forced movements. Occasionally the convulsive attacks bear a marked resemblance to hysteric conditions, or the general convulsions may begin with local spasms.

Vomiting.—Vomiting is a common symptom, being seen with greatest frequency in tumors of the posterior fossa. It often occurs without nausea and upon sudden changes of position.

Vertigo.—This occurs somewhat frequently, regardless of the location of the tumor. It is more apt to occur in tumors of the posterior fossa. The vertigo may be constant, or occur at intervals, and is frequently brought on by changes in position.

Optic Neuritis and Choked Disc.—These are the most important and most constant signs of cerebral tumor. The condition is always bilateral, although it is often more marked in one eye than in the other. As the result of the optic neuritis or choked disc optic atrophy eventually develops. A marked degree of optic neuritis may exist without impairment of the vision, the condition being revealed only upon ophthalmoscopic examination or examination of the visual fields, which latter shows a constriction and interlacing of the blue and red fields.

Pulse-rate.—The pulse-rate in cerebral tumor is usually slow, but may be irregular. In the terminal stage of a tumor the pulse-rate may become very rapid and intermittent.

Glycosuria and polyuria may develop during the course of a cerebral tumor.

The general symptoms are usually gradual in their onset, and progress steadily, with more or less rapidity, depending upon the location and rate of growth of the tumor.

Focal Symptoms.—Focal symptoms may be absent when the tumor is of very slow growth. They may likewise be absent in those cases in which the tumor is situated in some portion of the brain with whose functions we are not familiar, or disturbance of which does not give rise to symptoms. The focal symptoms depend upon the situation of the tumor, and arise as the result of irritation or destruction of a definite portion of the cerebral substance, and also indirectly owing to interference with the functions of a portion of the brain at a distance from the tumor. They may also occur as the result of interference with that portion of the brain affected by pressure or disturbance of its circulation. The focal symptoms are gradually progressive, and are usually limited in extent.

Focal Symptoms of Tumors of the Frontal Lobe.—Tumors affecting the first and second frontal convolutions may give rise to change in character, irritability, mental dulness, rapid mental fatigue, loss of self-control, and at times delirium and mild dementia. In other cases there are marked restlessness, a tendency to exaggeration, facetiousness, change of character and disposition, indifference, somnolence, or insomnia. In some cases in frontal lobe disease there is noticed a disturbance of the sense of equilibrium. Quite large tumors of the frontal lobes may exist without giving rise to any focal symptoms.

Tumors involving the third and frontal convolutions give rise to motor aphasia. If the left side of the brain is involved in right-handed individuals, or the right side of the brain in left-handed individuals, the speech disturbance, which is slight at first, gradually becomes more and more marked. At first the patient speaks slowly, and later on can speak only with difficulty. As the growth advances convulsions may occur, beginning first in the face and gradually extending to involve the muscles of the upper and lower extremities on the side of the body opposite to that on which the growth exists.

In those cases in which the tumor originated in or gradually involves the precentral convolution the focal symptoms are

those of a cortical epilepsy. Thus there occur tonic or clonic spasms of the muscles or convulsions, which may at first be limited in extent, and gradually extend. As time goes on the spasmodic seizures become more diffuse, and eventually the convulsions are replaced by paralysis. This paralysis may at first be limited in extent, but gradually becomes more and more extensive, progressing from a monoplegia to a complete hemiplegia.

The area first affected by the spasms and the order of their progression, and in later cases the order of involvement by the paralysis, are the most important localizing signs. Paresthesia, such as tingling, frequently precedes and generally follows these localized convulsive seizures.

The tendon reflexes are usually increased, and the superficial reflexes are decreased or lost on the side opposite to the tumor. Generalized convulsive seizures may occur in cerebral tumors without any direct involvement of the frontal lobe taking place.

Tumors situated in the parietal lobe are not always attended by distinctive local symptoms. If the tumor is situated in the anterior part of the parietal lobe, paresthesia occurs more or less persistently, and may be attended by spasmodic seizures. Deep tumors and large tumors in the parietal lobe give rise to disturbance of sensation upon the opposite side of the body, which may be accompanied by ataxia. Tumors in the posterior part of the parietal lobe may give rise to astereognosis. Tumors in the lower and posterior portion of the parietal lobe extending over into the anterior portion of the occipital lobe, if situated in the left hemisphere, in right-handed individuals give rise to alexia, or word-blindness. Tumors situated in the depths of the parietal lobe may press upon the optic radiations and cause hemianopsia.

Localizing Signs of Tumors of the Occipital Lobe.—Hemianopsia is often an early symptom that is entirely overlooked in tumors of the occipital lobe. Lesions of the lateral cortex of the left occipital lobe in right-handed individuals, and of the right lateral cortex in left-handed individuals, may give rise to a condition in which the individual sees, but fails to recognize

the objects seen. This condition is known as mind-blindness, and is closely allied to alexia.

Tumors of the temporal lobe involving the superior temporal convolution give rise to a condition known as word-deafness. In this condition the individual hears, but does not understand. Occasionally also tumors of the temporal lobe may cause disturbance of consciousness or convulsions, which are ushered in by auditory aura, such as ringing or whistling, or other sounds in the opposite ear. If tumors of the temporal lobe involve the deeper structures, they will compress the motor, sensory, and visual pathways, giving rise to paralysis, disturbance of sensation, and hemianopsia. Tumors involving the apex of the temporal lobe may give rise to disturbances of taste and smell.

Tumors involving the island of Reil give rise to symptoms by exerting pressure or irritation upon the neighboring structures, thus bringing about either motor aphasia, if they press forward; paralysis of the opposite half of the body, if they press inward; or sensory aphasia if pressure is exerted against the temporal lobe.

Tumors involving the optic thalamus cause awkwardness of movement, disturbance of muscle sense upon the side opposite to the growth, and sometimes abnormal movements, which may be athetoid in character. If the growth progresses, the posterior limb of the internal capsule becomes compressed, causing motor paralysis, disturbance of sensation, and hemianopsia.

Tumors of the corpus striatum do not give rise to focal symptoms, but as they increase in size they gradually compress or destroy the internal capsule, causing paralysis, disturbance of sensation upon the opposite side, and hemianopsia.

Tumors of the corpora quadrigemina cause paralysis of the external ocular muscles, sometimes pupillary paralysis, usually the oculomotor paralysis is not equal on both sides, nor is there always involvement of all the muscles supplied by these nerves. Nystagmus sometimes occurs; reflex automatic movements of the eyes are frequently interfered with. The gait resembles that of cerebellar involvement, being uncertain and staggering in character. Deafness and blindness may occur.

Tumors of the pineal gland may give rise to similar symptoms,

causing, in addition, paralysis of the fourth nerve. In some cases tumor in this region causes premature development of the sexual glands and exaggeration of the sexual impulses.

Tumors of the cerebellum occasion very early marked general symptoms. The focal symptoms of a tumor in this region are a staggering gait, with a tendency to fall always in a particular direction; intense vertigo, nystagmus, and headache, the last felt especially in the occipital region and in the neck, and sudden attacks in which unilateral convulsions, opisthotonos, and tonic contractions of the muscles of the body may occur. There is generally marked impairment of coördination and adiadokokinesis, which is more marked on the side of the tumor. Hypotonia and ataxia may also be caused by cerebellar tumors. In some cases abnormal positions of the head and body occur. If the tumor presses downward, the pons, medulla, and cranial nerves will be affected. Headache, vomiting, and vertigo are frequently intensified or brought about by changes in the position of the head.

Tumors of the cerebellopontine angle give rise to symptoms by compressing the pons or medulla, by compressing or irritating the cerebellar peduncles or cerebellum, and by compressing or irritating the cranial nerves situated in this region. As the result of compression of the pons or medulla, there is a paralysis or weakness of a spastic nature in the arm and leg on the side of the body opposite to the tumor. Delay in the transmission of sensory impulses or anesthesia may also exist on the opposite side of the body (Fig. 150).

The symptoms due to irritation or compression of the cerebellum or cerebellar peduncles are: a staggering gait, tending uniformly to go to one side, incoördination, sometimes ataxia, and adiadokokinesis, which is either limited or marked on the side opposite to that on which the tumor is situated.

The symptoms due to irritation or compression of the cranial nerves are: Intense pain of a neuralgic character in the distribution of the fifth nerve; this is usually followed by disturbance or loss of sensation along the course of this nerve. In some cases the neuralgic attacks do not precede the loss of sensation. In addition, there are paralysis or weakness of the external rectus

muscle; weakness followed by paralysis of the muscles of expression; attacks of pain in the ear, tinnitus, and deafness attended by vertigo; disturbance of swallowing; regurgitation of food through the nose; unilateral laryngeal paralysis; paralysis of one side of the tongue, causing defective articulation, etc.

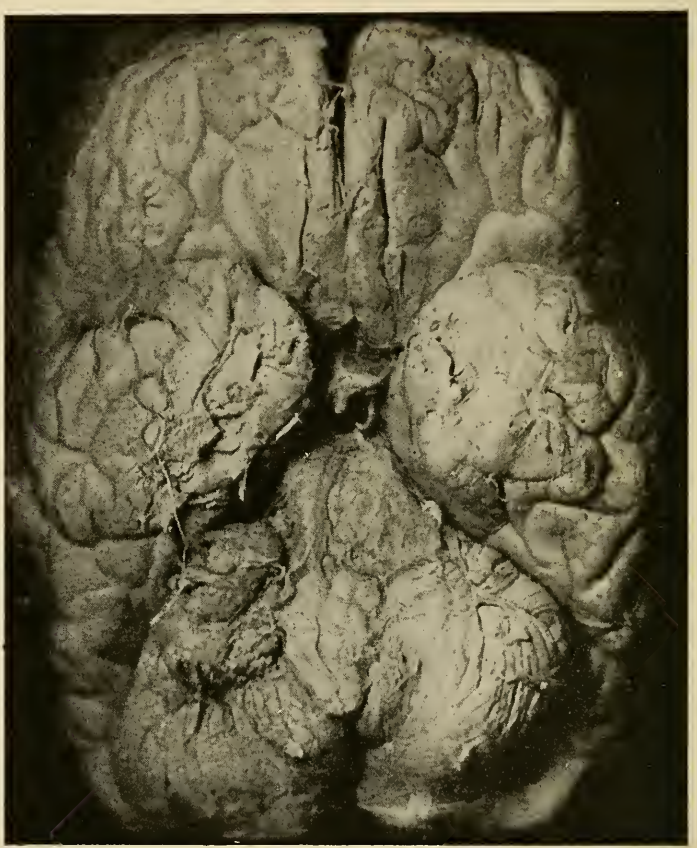


Fig. 150.—Brain showing tumor in right cerebellopontile angle.

Cranial nerve manifestations, when they occur, manifest themselves on the side of the body corresponding to that on which the tumor is situated.

Tumor of the cerebral peduncles is characterized by paralysis of the third nerve on the same side as the tumor, and weakness or paralysis of a spastic type upon the opposite side of the body. Tumors in this region also frequently give rise to abnormalities

of movement, causing tremors, which may be intentional in character or resemble the tremor of paralysis agitans.

Oculomotor paralysis, tremor, and hemiplegia upon the side of the body opposite to the oculomotor paralysis constitute what is known as Benedikt's syndrome.

Tumors of the third ventricle are especially prone to cause drowsiness and a tendency to fall into a comatose condition. Tumors of the pons cause paralysis of the third, fifth, or sixth nerves, with pain, disturbance of sensation, anesthesia, trophic disorders in the distribution of the fifth nerve, sometimes grinding of the teeth on the side of the tumor, with weakness or paralysis of a spastic nature upon the opposite side of the body. Tumors in the medulla cause weakness or paralysis in the distribution of the ninth, tenth, eleventh, or twelfth nerves on the side with the tumor, together with weakness or paralysis of a spastic type upon the opposite side of the body.

In those cases in which the tumor is sufficiently large to compress the deeper structures of the pons, crura, or medulla, sensory disturbances may be added to those of the motor type, and if the tumor increases in size, so as to involve these structures more extensively, weakness and paralysis finally attack the unaffected side of the body.

Tumors occurring in the hypophysis cerebri give rise to three sets of symptoms: First, the general symptoms of an intracranial tumor; second, those due to interference with the internal secretions of this gland; and third, symptoms due to involvement of the structures in the immediate vicinity of the gland.

The first set of symptoms has been previously enumerated. The second set of symptoms includes gigantism or acromegaly, if the gland is stimulated to increased activity; adiposity, with increased tolerance for carbohydrates, with or without genital hypoplasia if the glandular secretion is diminished or interfered with. The third set of symptoms is the result of pressure or destruction of the surrounding structures; thus if the optic tracts or chiasm are pressed upon or destroyed, bitemporal hemianopsia occurs, and frequently the third and fourth nerves are affected; exophthalmos may occur if the cavernous sinus is pressed upon or if the tumor invades the orbit; if pressure is made

on the crura, hemiplegia will occur, and in some cases the sense of smell is lost.

Course.—A cerebral tumor may exist for a considerable length of time without giving rise to any definite symptoms. It may manifest its presence more or less suddenly, particularly in gliomatous tumors, the first evidence of its existence being a sudden apoplectic seizure due to hemorrhage into the growth. Other tumors manifest themselves by symptoms pointing to irritation of a localized cerebral area, whereas others first give rise to general cerebral symptoms. As time goes on, in some cases more rapidly than in others, there is a gradual increase and progression of symptoms. The disease may run a more or less rapid course, a fatal termination occurring within six or eight months, but the usual duration is about three years. Some tumors are present for many years, and in some cases the growth is discovered only after death from some intercurrent disease. The location and character of the growth influence the rapidity with which the symptoms develop, and upon these is dependent the duration of life after the first symptoms have manifested themselves. Tubercular growths, glioma, and carcinoma usually develop rapidly; sarcoma, syphilitic growths, and fibroma may develop more slowly; on the other hand, tubercular growths may develop very slowly and syphilitic growths very rapidly. Tumors situated in the posterior cranial fossa and those involving the basilar ganglia and brain-stem give rise to a more rapid development of symptoms. Growths involving the medulla usually cause a rapid fatal termination.

Prognosis.—The prognosis of cerebral tumors depends to a great extent upon the character of the growth, the possibility of locating it, its accessibility to surgical intervention, and upon the patient's general physical condition and whether the diagnosis is made early or late.

Gliomatous tumors, carcinoma, and diffuse sarcoma, owing to the fact that they cannot be entirely eradicated, are not amenable to surgical treatment, and consequently the outlook in these tumors is hopeless. Encapsulated sarcoma, when situated in a position that is surgically accessible, may be removed, with

more or less complete recovery, this depending upon the amount of brain tissue that has been destroyed at the time of the operation. Tubercular growths are sometimes arrested spontaneously. As a rule, when an effort is made to remove them surgically there is subsequent infection of the meninges, with recurrence of the growth or the development of a tubercular meningitis.

Syphilitic growths are generally amenable to antisyphilitic treatment. Cysts, if situated in operable areas, and if they are not secondary to glioma, can very frequently be removed by drainage and excision of the cyst-wall.

Diagnosis.—When the presence of a cerebral tumor is suspected, it is necessary, in order to confirm the diagnosis, to exclude nephritis, meningitis, general paresis, cerebral abscess, and lead-poisoning.

Tumor is differentiated from *nephritis* by careful examination of the urine, the variability of symptoms in nephritis, the presence of a neuroretinitis rather than an optic neuritis, usually rapid pulse with high arterial tension and the onset of the symptoms, which may be either very rapid or extend over a period of years. In tumor the onset of the symptoms is gradual, and the condition progresses over a period of from a few months to one or two years; there is more apt to be an optic neuritis than a neuroretinitis, and the urine may be negative, the pulse slow, the blood-pressure not necessarily high, etc.

From *meningitis* tumor is distinguished by the absence of temperature in the latter condition, by the rapid, irregular pulse in meningitis, the absence of cervical rigidity in tumor, the presence of leukocytosis and the more acute onset in meningitis, and by the results of examination of the cerebrospinal fluid.

Cerebral tumor is differentiated from *general paresis* by the presence, in the latter, of optic atrophy rather than optic neuritis, pupillary disturbances which are bilateral, the examination of the cerebrospinal fluid yielding a positive Wassermann reaction, with an increased cell-count, and by the presence of disturbances of memory and of speech which are characteristic of paresis.

From a slowly progressive *cerebral abscess* tumor is sometimes distinguished only with great difficulty. In these cases the history of a possible focus of origin of the abscess is very im-

portant. As a rule, the progression of symptoms is accompanied by marked temperature disturbances; leukocytosis is usually present, and there are elevations of temperature with exacerbation of symptoms and a subnormal temperature in the intervals.

From *lead-poisoning* tumor is differentiated by the examination of the blood, the history of exposure to lead-poisoning, and the intermittent progress of the symptoms.

If the presence of a cerebral tumor has been positively determined,—and this can usually be done in about 85 per cent. of the cases,—it is necessary to locate the tumor, and to decide whether or not it can be removed. The character of the growth must also be determined, and whether or not it is single or multiple. The location of the growth is determined from the focal symptoms and the history of the progress of the general and focal manifestations. In some cases in which the growth is on or near the surface of the brain, the bone over the growth may be eroded and the tumor may be palpable. In some few cases there are pain and tenderness on percussion on the surface over the growth. Whether or not the tumor is removable surgically is dependent upon the location and the character of the growth, and on whether it is single or multiple. Only those growths that displace or compress, but do not infiltrate, the brain tissue are surgically removable. Multiple growths and very extensive diffuse growths cannot be removed. Tumors involving the ventricles, the basilar ganglia, or other portions of the brain not accessible to surgical intervention are inoperable. The character of the growth can be diagnosed only approximately before operating: one must take into consideration the age of the patient, the progress of the symptoms, the presence of syphilis, tuberculosis, or malignant growths in other portions of the body, trauma, etc.

In general, the diagnosis of tumor of the brain rests upon a history of more or less persistent headache, vomiting, and vertigo, extending over a considerable period, and the presence of choked disc, and in some cases of distinctly progressive symptoms pointing to irritation or destruction of a local cerebral area. The examination of the cerebrospinal fluid, and the general physical examination of the individual, and in some cases in

which the tumor is accompanied by bone changes, such as tumors involving the sella turcica, a radiographic examination, are all aids to correct diagnosis.

Treatment.—In all cases in which there is a progressive optic neuritis a subtemporal decompression operation should be performed. In those cases in which syphilis is suspected as the underlying cause, a thorough course of antisyphilitic treatment should be instituted. The patient's general physical condition should receive attention. Pain should be relieved by the administration of anodynes. Vomiting and convulsions are usually best treated by rest and the adminstration of the bromids. A light nutritious diet should be ordered, and tonics should be given to conserve the patient's general strength. In those cases in which the growth is localized and is situated in a region of the brain that is accessible surgically an effort should be made to effect its removal. In the hands of those who are specially trained in neural surgery operative intervention is a comparatively safe procedure.

PARALYTIC DEMENTIA (GENERAL PARESIS; GENERAL PARALYSIS OF THE INSANE)

This is a disease of the brain characterized chiefly by progressive weakness of the mind and of the body.

Etiology.—Heredity, intellectual overwork, trauma, worry, chronic poisoning by alcohol, etc., are all important factors in the production of this disease, but the principal underlying cause in practically all cases is syphilis. Males are more frequently affected than females. The disease usually manifests itself between the thirtieth and the fiftieth year, and occurs only rarely after the sixtieth year. Frequently the condition develops in the very young, when it is believed to be due to hereditary syphilis. The disease is more prevalent in large cities than in rural communities, and is also more apt to affect those engaged in intellectual pursuits or those whose occupation requires constant mental alertness than those who are not exposed to mental strain. In addition to syphilis, other chronic toxemias, such as alcohol and tobacco, and gastro-

intestinal disturbances, occupy an important position in the production of the condition. Trauma, mental strain, worry, physical fatigue, acute intoxication, physical and sexual excesses, exhausting diseases, emotional disturbances, such as fright, joy, etc., are also frequent etiologic factors.

Pathology.—The pathologic changes occur mainly in the brain. The spinal cord is usually involved also, and sooner or later pathologic changes appear in practically all the organs of the body. The brain is generally decreased in size; the convolutions appear atrophied; the fissures are wider and deeper. Atrophy affects especially the frontal lobe, although the entire brain may be affected. The pia mater is generally firmly adherent to the brain, and cannot be removed without particles of the cortical substance adhering to it. The meninges are thickened and often opaque; the ventricles are somewhat dilated; the endothelial linings show granular and inflammatory changes; the arteries exhibit sclerotic changes; the cerebrospinal fluid is generally increased in amount, and contains a larger number of cellular constituents than normally. Microscopically there is seen a degeneration and atrophy of the cells and fibers of the cerebral cortex which is exceedingly diffuse in distribution. Degenerative changes are also discovered in the basilar ganglia, brain axis, and in the spinal cord. Gray degeneration of the optic nerve and sometimes of the other cranial nerves occasionally occurs. There is a proliferation of glial tissue throughout the cortex, being most marked around the blood-vessels. The various organs of the body, such as the liver, kidney, heart, etc., show changes of a chronic inflammatory nature.

Symptoms.—This disease develops more or less insidiously, and in the early stages is characterized by symptoms that are more or less indefinite in character. During this period the patient may be sleepless and irritable, and complain of vague pain and inability to concentrate the thoughts or to carry on prolonged mental work. There may be digestive disturbances, loss of appetite, depression, headache, and a general feeling of illness, or these symptoms may be absent and in their stead it may be noticed that the individual's memory is inaccurate,

and he may manifest a peculiarity of speech; in writing, words are frequently misspelled, syllables or entire words being omitted, or there may be a reduplication of words or syllables. There is frequently observed, early in the disease, indifference to the higher sentiments, lapses from the proprieties, and disregard or failure of interest in important matters. In other cases the patient becomes depressed and indifferent, easily upset, displays weakness of judgment, and is unduly susceptible to external influences. Other patients become active, restless, egotistical, and easily excited, and show a marked tendency toward exaggeration and extravagance. These patients frequently display tendencies to sexual and alcoholic excesses. Their judgment is poor, the higher feelings become blunted, and they exhibit a marked tendency to disregard the rights of others. In the early stages the disease is characterized principally by vague subjective physical symptoms and by gradual weakness of the mental powers; judgment is interfered with, and the finer feelings become dulled. There is a loss or decrease of self-consciousness, and the conduct becomes less restrained. Usually there are physical objective signs that enable one to suspect the underlying condition; for example, weakness or slight paralysis of one side of the face; transitory ocular paralysis; unequal pupils or pupils that are abnormally contracted or fail to react to light. The tendon reflexes may be markedly diminished, lost, or increased, and there may be fibrillary tremor of the facial muscles or the tongue upon voluntary movement, slight difficulty in articulation, and epileptiform or apoplectiform attacks. Examination of the blood and cerebrospinal fluid, even at this stage, will in the vast majority of cases reveal evidence of the presence of a syphilitic infection (Fig. 151).

As the disease progresses, it assumes one of three types: the demented type, the depressed, or the exalted type. In any one of these three types hallucinations may be present; in the demented type there is a progressive failure of memory for both remote and recent events, for the passage of time, and the patient may fail to recognize himself in his surroundings and frequently mistake the personality of those with whom he comes into contact. Often he becomes indifferent to his surroundings,

but at times there may be frequent outbursts of anger and irritability. In the depressed type the individual becomes despondent, pessimistic, and is unable to reach a conclusion. He is frequently apprehensive of his own condition. Later his ideas are of a self-accusatory character, his judgment becomes weakened, and suicidal attempts are common. In addition to the depressive features, these patients show disturbances of memory, failure of judgment, and moral and ethical indiffer-

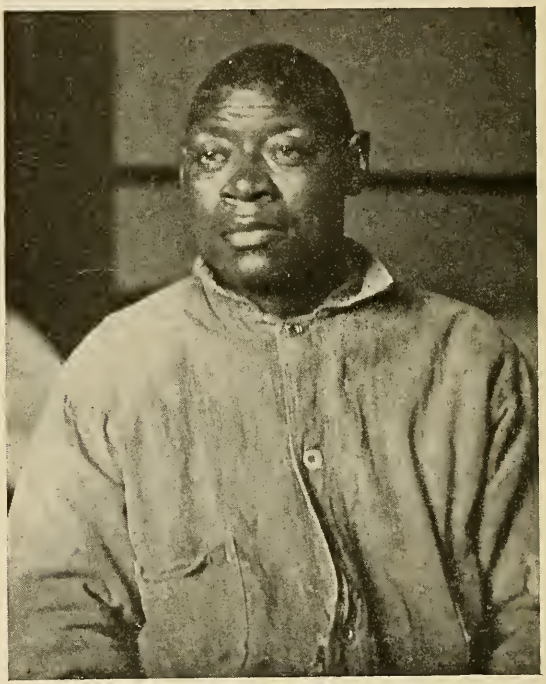


Fig. 151.—General paresis, early stage (Bay View Hospital).

ence. In the exalted type the patient becomes expansive, egotistical, and restless, and rapid changes of emotion, from marked irritability and anger to happiness and good-fellowship, occur. These patients are especially prone to sexual and alcoholic excesses. As the result of their weakened judgment they frequently undertake all sorts of absurd and senseless schemes. In conjunction with these mental manifestations hallucinations may occur.

The second stage is characterized by progressive failure of

memory, increased weakness of judgment, emotional disturbances, and marked obtunding of the ethical, esthetic, and moral feelings. Delusions of a depressive type may also be present. The objective physical signs of the first stage are intensified. The speech disturbances are characteristic—there is a duplication or elision of syllables and letters; this is well shown in the efforts to pronounce test phrases, such as "Third German riding artillery brigade," "Massachusetts," "Methodist-Episcopal," "immovable," etc. Artillery is frequently pronounced artilry, or artillilery, brigade, bigrad, etc.

As the disease progresses the difficulty in pronouncing words and in remembering phrases increases. The speech is usually attended by a marked fibrillary tremor of the face and tongue. The handwriting also shows marked disturbances—words are omitted, words, syllables, or even entire sentences are repeated, and very frequently there is a fine tremor of the fingers, accompanied by a coarse tremor of the hands which evidences itself upon voluntary movement.

In at least 95 per cent. of cases pupillary disturbances are present. The Argyll Robertson pupil is present in most cases, and there are also frequently inequality of the pupils, extreme pupillary contraction, irregularity of pupillary outline, and sluggish pupillary reaction to light.

The deep reflexes are generally increased. In those cases in which the spinal symptoms are marked the deep reflexes may be diminished or lost. There may be marked difference of the deep reflex responses of the two sides of the body. As the disease progresses there is a disturbance in the control of the rectal and vesical reflexes.

Sooner or later motor disturbances occur. Apoplectiform or epileptiform seizures or progressive weakness develop, and attacks of unconsciousness and of vertigo are also frequently observed. Weakness of intellect and transitory or permanent cranial nerve paralysis occur in both the first and the second stage of the disease. In the early stages of the affection there is often a loss of weight, but in the second stage, when the dementia is once under way, there is often a gain of weight. Hallucinations and illusions frequently occur in the second

stage of the disease, and bear a distinct relation to the emotional state. In the second stage of the disease remissions may occur, and an apparent improvement in the patient's mental and physical condition take place. In some cases the improvement attains so marked a degree that it is almost impossible to discover evidences of mental disease. The physical signs, of course, remain permanently present, and sooner or later there is a recurrence of the symptoms, or without a remission in the symptoms the patient may enter into the third or terminal stage.

In the third stage the evidence of dementia becomes more marked, and the speech disturbance is more pronounced, until finally speech becomes a mere mumbling of sounds. Progressive inanition occurs, trophic disorders manifest themselves, and the individual becomes neglectful in his habits and finally helpless and bedridden. Grinding of the teeth becomes marked, and there is difficulty in swallowing. Attacks of coughing and choking occur, and finally death follows as the result of pneumonia, septicemia, or exhaustion. Some patients die before this stage is reached, the fatal termination being due to some intercurrent condition or to apoplectiform or epileptiform attacks.

Course.—This disease is usually insidious in its onset, existing frequently for weeks or months before it is recognized. It runs a slowly progressive course. At times there are acute exacerbations of symptoms, these being particularly apt to occur after a paralytic attack. Remissions not uncommonly occur with an almost complete disappearance of the subjective symptoms. These remissions may last for from a few days to six months, rarely longer.

In the greater number of cases the disease runs its course within three or four years; in exceptional cases death occurs within a few months, and in very few cases the illness may last for ten years or more. Those cases showing dementia and those in which spinal symptoms are marked usually run a slower course.

Prognosis.—Prognosis is extremely unfavorable. Recently a few cases of general paralysis have been reported in which

cure followed the use of intravenous and intraspinous medication. As yet, however, the number of patients so treated has not been sufficiently large to warrant a statement as to the efficiency of this form of treatment. Even if it were capable of arresting the progress of the disease, it cannot restore the brain tissue that has already been destroyed. At best, one can expect good results only in those cases that are diagnosed before pronounced symptoms have occurred.

Diagnosis.—In the early stages of the disease general paresis may be confused with neurasthenia, cerebral arteriosclerosis, and cerebrospinal syphilis.

Neurasthenia	General Paresis
Pupillary Disturbances	
Pupils may be dilated or contracted, but react to light and are not irregular in outline.	May be dilated or contracted, react sluggishly or not at all to light, and may be irregular in outline.
Deep Reflexes	
Usually active and equal.	May be increased, decreased, lost, or unequal.
Tremor	
Fine rapid tremor of the fingers and hands.	Fine fibrillary tremors involving the trunk and facial muscles.
Memory	
Patient complains of disturbance of memory, which really does not exist.	Patient usually shows true defects of memory.
Complaints	
Complains at length of many physical and mental symptoms, and is apprehensive of the presence of organic physical disease.	Frequently has no complaint; at times there is marked variability in his complaint; often the emotional tone is not in accord with the complaint.
Actions	
No changes in the moral and higher feelings.	A decrease of the finer and moral feelings, evidenced by actions that are not in accord with the previous life of the patient.
Age	
The first attack occurs more frequently before the thirtieth year.	Occurs more frequently after the thirtieth year.

General Arteriosclerosis	General Paresis
Age	
Generally after the fiftieth year.	Generally between the thirtieth and fortieth year.
Pupillary Disturbances	
Pupils may be contracted or dilated, unequal, but react to light.	Pupils contracted or dilated, unequal or irregular, fail to react to light.
Tremor	
Involves the extremities and head; is intentional in character.	Fine fibrillary tremors of facial muscles, involving also the trunk.
Memory	
Disturbed more for recent events, and only in terminal stage for remote events.	Diffuse disturbance of memory, both remote and recent.
Examination of the Cerebrospinal Fluid	
Negative Wassermann and cell count.	Positive Wassermann, increased cell count, and positive globulin reaction, etc.

Cerebrospinal Syphilis	General Paresis
Headache	
More or less severe and constant.	Infrequent and rarely persistent.
History of Syphilitic Infection	
Of recent date.	Five to twenty years preceding.
Pupillary Disturbance	
May be absent, or present in one eye; or there may be immobility to both light and accommodation.	Argyll Robertson pupil.
Cranial Nerve Paralysis	
Frequent, and of rapid onset.	Infrequent; of slower onset, and transitory.
Speech Disturbance	
Usually not present, or if present, of an aphasic character.	Speech disturbance generally present, with a characteristic elision or reduplication of syllables and words.
Mental State	
Stuporous states frequently present.	Rarely present.
Portion of Brain Involved	
Symptoms point more to basilar involvement.	Symptoms point chiefly to cortical involvement.

The diagnosis of general paresis, as a rule, does not present much difficulty if one takes into consideration the change of character and of disposition of the individual, together with the physical signs of the disease.

Some cases of general paresis occur, however, which, in the early stages, present typical neurasthenic symptoms, and in which there is an absence of the physical signs. In these individuals a mistaken diagnosis may be made, the true state of affairs not being revealed until a subsequent period, when the patient's actions or the discovery of physical signs clear up the diagnosis. One should always look with suspicion on previously healthy individuals that present neurasthenic symptoms for the first time after the age of thirty-five or forty. There also occur cases in which the differentiation between cerebrospinal syphilis and general paresis is absolutely impossible, the subsequent course of the disease and the response to treatment being the only true guide to correct diagnosis.

At times paresis may first manifest itself by a sudden maniacal outbreak, and under these conditions it must be differentiated from the excited phase of some other form of mental disease. In these cases the history is important. The excitement is rarely so marked or so sudden in other mental disorders as it is in paresis, and the presence of the physical signs of paresis and the examination of the cerebrospinal fluid will usually enable one to reach a correct conclusion. If paresis manifests itself first in apoplectic or epileptic attacks, difficulty may be experienced, first, in differentiating the patient's condition from vascular cerebral disease, epilepsy, and other organic cerebral diseases. Under these conditions the subsequent history of a gradually progressive disease, the presence of physical signs of paresis, the examination of the cerebrospinal fluid, and a history of the preceding physical and general state of the patient will confirm the diagnosis.

Treatment.—This is largely prophylactic, and consists of proper and prompt treatment of syphilitic infection. In those who have had this infection, the mode of life should be as comparatively free as possible from mental and physical strains, and excesses of all kinds should be avoided.

As soon as the diagnosis is made, the patient's family should

be notified of the danger that threatens the patient because of his disturbance of judgment, the likelihood of his squandering or wasting his means, and the possibility of trouble because of his changed conduct. The individual should be placed under the necessary legal restrictions, and should be guarded and protected from harm. In most cases committal to an institution is advisable.

The patient's general health should be looked after. Rest in bed is frequently beneficial. When depression exists, opium frequently gives relief, and during the periods of excitement the administration of hyoscin, together with rest in bed and the application of warm packs tend to alleviate the condition. Restlessness and insomnia can frequently be relieved by the administration of bromids. In the later stages of the disease care should be taken in feeding of the patient so as to prevent particles of food from entering the lungs. The development of bed-sores should be prevented if possible, and the bowels and bladder should receive careful attention.

Naturally, in each case of paresis the question arises whether or not the condition is amenable to antisyphilitic treatment. In my opinion every paralytic should receive a thorough course of treatment and the effects thereof be noted. This, of course, applies only to those cases in which the diagnosis is made in the first or second stage of the disease, and whose general condition warrants the administration of vigorous antisyphilitic treatment. Salvarsan should be given intravenously in appropriate doses at intervals of two or three weeks. Simultaneously with the salvarsan should be given intraspinous and intraventricular injections of salvarsanized serum containing a small dose of mercuric chlorid. In the intervals between the administration of salvarsan, and beginning three days after the injection and stopping two days before an injection, mercurial inunctions should be made daily, and potassium iodid in moderately large doses should be administered internally. The effect of this treatment upon the blood and spinal fluid should be noted by proper serologic and chemical examinations. It is the experience of those who have used this form of treatment that some few patients are improved, that the general condition of most cases is rendered better, and that a few have been cured.

DISEASES OF THE BRAIN AND SPINAL CORD

SYPHILIS OF THE NERVOUS SYSTEM

About 10 per cent. of all persons who become infected with syphilis show involvement of the nervous system at a later date. The nervous system, it would seem, is more apt to be involved in those cases that showed the mildest symptoms at the onset. Thorough treatment in the first and second stages has not always prevented involvement of the nervous system at a subsequent period. At the present time, as the result of more recent methods of treatment, this sequel, it is hoped, may be avoided.

When syphilis involves the nervous system, it does so in one of several ways: First, the meninges, superficial tissues, and perivascular spaces may be invaded by gummy exudative material: this is particularly apt to occur within a few months after infection. Second, there may be cellular infiltration of the membranes, brain, spinal cord, or nerves, which is of a more or less chronic nature. Third, the nervous system may be so affected as to give rise to conditions that were formerly called parasyphilitic, but which we now believe to be due directly to the presence of the Spirochæta pallida or its products. Fourth, the blood-vessels of the central nervous system may be affected as the result of the activity of the Spirochæta pallida, or of substances formed during the life of this organism, bringing about either an endarteritis, with consequent decrease in the lumen of the vessel, or degenerative changes of the vessel-walls which may either interfere with the proper cerebrospinal circulation, or, by weakening their walls, predispose them to rupture. Fifth, in many cases there is a combination of two or more of the preceding conditions.

The symptoms of syphilitic affection of the nervous system may be conveniently grouped as follows: First, those symptoms

that are caused by syphilitic disease of some structure adjacent to the nervous system, which, by extension, interferes with or affects the functions of the nervous organism; such are severe head pain, caused by a syphilitic periostitis; pressure on nerves or the spinal cord, caused by syphilitic disease of the surrounding structures, etc. The cause of these symptoms can usually be discovered as the result of proper physical and laboratory examinations.

A second group of cases, in which the manifestations are due to syphilitic exudation about the blood-vessels, in the meninges of the brain, and in the spinal cord, are usually observed soon after the initial infection. This exudate occurs as a diffuse, transparent, sticky mass, or it may be localized, forming a tumor. Such tumors are often multiple. This exudate is usually deposited rapidly, and produces pressure upon both the neural and the vascular tissue in its immediate neighborhood, causing suspension of function of these tissues. The symptoms that arise as the result of the presence of this substance depend upon its location. There are generally headache, which is worse at night, restlessness, and general discomfort. To these there are added more or less suddenly disturbance of the functions of the brain, such as epileptiform attacks, disorders of consciousness, the various cranial nerve palsies, optic neuritis, partial or complete hemiplegia or hemianesthesia, etc. In other cases there may be a gradually increasing headache, accompanied by increasing mental dulness, to which may be added disturbance of the pupillary reaction, optic neuritis, etc. Other cases are seen that present only the symptoms of tumor of brain. In those cases in which the blood-vessels are principally involved the symptoms that arise are those referable to interference with the cerebral circulation.

In those cases in which the exudate involves the spinal cord, there will be pain in the back, disturbances of sensibility on the trunk and lower extremities, and perhaps sharp shooting pains and girdle sensation, disorders in the control of the bowels and bladder, together with more or less weakness of the extremities, depending upon the location of the lesion.

The more extensive the involvement of the meninges, the more severe will be the pain and rigidity; and the more marked the involvement of the cord substance itself, the more prominent will be the symptoms that can be referred to the interference with the transmission of impulses upward and downward through the cord.

In those cases in which there is a cellular infiltration into the membranes or nervous tissue itself, the symptoms are usually of slow onset, the condition running a chronic course. If this infiltrative process chiefly involves the membranes, the most prominent symptoms are those of a chronic meningitis; if, on the other hand, the nervous tissues are principally involved, symptoms appear that may resemble general paresis, multiple sclerosis, diffuse myelitis, and bulbar paralysis.

The so-called parasyphilitic diseases—tabes dorsalis and general paresis—are described in other sections of this volume.

The symptoms that occur as the result of syphilitic disease of the blood-vessels of the brain and spinal cord differ in no way from those symptoms that have been described in previous chapters, dealing with vascular diseases of the brain and cord.

Hereditary syphilitic diseases of the nervous system usually appear during the first period of life, but frequently may not become evident until puberty. The pathologic changes are practically identical with those that occur in acquired syphilis. In hereditary syphilis the symptoms are prone to be more severe and extensive than in acquired syphilis, and inhibitions in the development of the neural tissue are frequent.

The diagnosis of syphilis of the nervous system is usually readily made; in doubtful cases a Wassermann test of the blood and spinal fluid, the Noguchi and other tests of the spinal fluid, and the cell count of the spinal fluid will generally enable one to reach a correct conclusion.

Prognosis.—Under proper treatment the prognosis as regards cure of the syphilitic process is very good: those cases in which the symptoms are due to exudative syphilitic processes almost always clear up; those in which the symptoms are due to an infiltrative process usually improve, but some permanent symptoms may remain. The parasyphilitic affections can now be

greatly improved, and the course of the process in many cases arrested. Those symptoms that arise as the result of destruction or degeneration of a portion of the nervous tissue remain permanently. Cases that are due to syphilitic affection of the blood-vessels may or may not be relieved by treatment, depending altogether upon the character of the vascular involvement. Patients in whom the perivascular tissues are affected may be greatly benefited by treatment. In those cases in which the intima is involved, with the production of an endarteritis, the process may be arrested and the symptoms alleviated, but not entirely eliminated. Symptoms due to hereditary syphilis offer the same prognosis as to recovery under treatment as do those due to acquired syphilis, with the exception of those manifestations that are due to arrest of development of the nervous tissue.

Prophylaxis of Syphilis of the Nervous System.—This end is best subserved by beginning the thorough treatment of syphilis as soon as the diagnosis is made. In addition to the usual methods of treatment, which will be described further on, in every case of syphilitic infection, at some time during the course of treatment, and certainly before the patient is discharged as cured, spinal puncture should be made and the spinal fluid carefully examined. If there is evidence of involvement of the nervous system, even in the presence of negative findings in the blood, the proper treatment, described below, should be administered.

Treatment.—The treatment of syphilis of the nervous system has as its basis the complete eradication of the syphilitic virus from the blood, the various organs and parts of the body, and the central nervous system. There are certain peculiarities of the nervous system that must be taken into consideration when treatment is directed toward the cure of syphilitic diseases of this portion of the body: the first is the difficulty with which medicinal substances can be made to penetrate the deeper portions of the brain and spinal cord; second, the peculiar fact that the choroidal plexus practically does not allow the passage of metallic substances from the blood into the cerebrospinal fluid.

At the present time, from the large number of drugs at our disposal, mercury and arsenic seem to stand out preëminently as the two substances on which we must rely in order to hope to effect a cure of syphilis. The iodids, although very useful, and I might say almost indispensable, for the relief of symptoms, certainly cannot be classed among the drugs to be employed for the eradication of the Spirochæta pallida. Mercury can be introduced into the system by way of the mouth, by inhalation, injection, and inunction, and at times it may be necessary to employ several of these routes. As a rule, however, the best results, with the fewest disagreeable manifestations, are obtained by means of inunctions. Salvarsan, of course, is now almost universally given by the intravenous method.

It has been my experience that syphilitic disease of the nervous system yields most promptly to the following line of treatment, which should always be controlled by the Wassermann examination of the blood and spinal fluid, including the cell count and the globulin reaction of the spinal fluid. The patient is given a dose of salvarsan appropriate to his age, and two days later I advise the use of mercurial inunctions daily. This treatment is continued for thirty days, unless contraindications arise, when salvarsan is again given, followed by the mercurial inunctions. This proceedure is continued until the symptoms have disappeared and the Wassermann examination of the blood is negative. Examinations should be made once every six months for several years. In those cases that show a positive Wassermann, increased cell-count, and globulin in the cerebrospinal fluid the treatment consists of inunctions of mercury, the intravenous injection of salvarsan, and the intraspinous and at times the intraventricular administration of autosalvarsanized and mercurialized serum.

The following is an outline for the treatment of a patient affected with syphilis of the nervous system who has positive findings in both blood and cerebrospinal fluid: If the patient is an adult, he is given six-tenths of a gram of salvarsan intravenously; then, following the technic of Swift and Ellis, a 60 per cent. autosalvarsanized serum is prepared; to about 30 c.c. of this solution I usually add one-fiftieth of a grain of mercuric

chlorid. A spinal puncture or ventricular puncture is made, a proper amount of cerebrospinal fluid is withdrawn, and the autosalvarsanized and mercurialized serum is slowly injected into the subdural space or into the ventricle. Following this procedure there may be some reaction, consisting of general malaise, headache, vomiting, diarrhea, pain in the back, pain in the legs, and sometimes bowel and bladder disturbance. This reaction may last for one or two days, and vary in severity. On the third day after this treatment the patient should receive an inunction of one dram or more of mercurial ointment, repeated daily, care being taken to avoid salivation. Potassium iodid in suitable doses is also administered. After three or four weeks the mercury and iodid are discontinued for two days, and an examination of the blood and spinal fluid made. If these are still positive, the same course of treatment is repeated, and continued until both blood and spinal fluid become negative. After this a Wassermann test of blood and spinal fluid should be made at intervals of six months for two years. In those cases in which the spinal fluid becomes negative before the blood, the intraspinous injections may be discontinued, but the salvarsan, mercurial inunctions, and potassium iodid should be continued. As long as the spinal fluid shows positive findings it is necessary to continue the treatment as directed, giving salvarsan intravenously, salvarsanized mercurialized serum intraspinally or intraventricularly, and inunctions daily in the intervals between the administration of the salvarsan and the giving of the potassium iodid if symptoms are present.

If, after the treatment, there is no reappearance of pathologic findings in the blood or cerebrospinal fluid, we may safely conclude that a method has been used that promises a complete cure of the syphilitic infection. It is, of course, too much to expect that any form of treatment will restore the original function of those structures that have been destroyed as the result of the disease: the most we can expect is that the method of treatment will permanently eradicate the cause of the disease.

MULTIPLE SCLEROSIS (DISSEMINATED SCLEROSIS)

Multiple sclerosis is a disease that begins in youth. It is characterized by the production, in the central nervous system, of small areas of sclerotic tissue that are irregular in distribution.

Etiology.—This disease usually manifests itself between the tenth and the twenty-fifth year, although no age is exempt. Some cases occur early in childhood, and others do not manifest themselves until later in life. Cold, injury, infectious diseases, and toxic agents are all believed to predispose to, or act as exciting factors of, the disease. It is now generally believed that this disease is a sequel of one of the acute infectious diseases, such as typhoid, influenza, whooping-cough, acute articular rheumatism, scarlet fever, etc. It has also been observed to follow chronic intoxication with metallic poison. The disease has no connection with syphilis. By some observers, a congenital maldevelopment of the nervous system is regarded as the direct cause, while the infectious diseases, trauma, toxemia, etc., are considered to be the exciting causes. In many cases no cause is discoverable.

Pathology.—Macroscopically, there can be seen scattered over the surface of the brain, medulla, and spinal cord areas of varying sizes and shapes and grayish-blue in color. These are especially prominent on transverse section. They vary in size: some are so large as to permeate almost the whole thickness of the cord or medulla, or they may be so small as to be recognized only microscopically. These areas may principally involve the cord, medulla, or brain, or all three of these structures may be involved more or less equally. They also occur frequently in the optic tracts and chiasm. These foci are usually sharply defined, and may assume any form. They are generally of a firmer consistency than the normal nerve tissue, and frequently there is some infiltration of the tissue outside of the focus.

Histologic examination of the foci shows disintegration of the myelin sheaths of the fibers, with persistence of the axis-cylinder process and an increase of the interstitial tissue. The foci in the gray matter affect the cells to a very slight extent. Fre-

quently there occur the formation of new blood-vessels and occlusion of others. Owing to the fact that the axis-cylinder process and the cells are spared in these sclerotic patches, there is almost a complete absence of secondary degeneration. It would seem as though a toxic substance, exercising a selective activity on the myelin sheaths, caused their destruction, together with a proliferation of the neurogliar elements, and spared the cells and the axis-cylinder processes. No explanation can be given for the peculiar and irregular distribution of the foci.

Symptoms.—There are three symptoms that occur in practically all cases. They are intention tremor, nystagmus, and disturbance of speech. When one takes into consideration the fact that the early symptoms of this disease may be due to involvement of the spinal cord, the brain-stem, or some other portion of the nervous system, and that in some cases there is complete intermission of or disappearance of certain symptoms for many months, with their reappearance at some later date, it can be seen how difficult it is to describe accurately the early stages of this disease. The symptoms are usually very gradual in their development: the patient may complain of numbness and weakness in the legs, which slowly increase until the gait becomes spastic, ataxic, or of the cerebellar type. In some cases little or no weakness develops, but the legs become extremely stiff, with increased knee-jerks, ankle-clonus, and the Babinski sign. Decrease or absence of the superficial reflexes often occurs early. Usually, when the patient walks the entire body sways. In some cases, along with the spastic impairment of gait there is also marked uncertainty, so that there is danger of falling. Active movements of the limbs, head, or trunk are usually accompanied by tremor.

Tremor is absent during rest, but when the patient attempts to perform some voluntary act, from the beginning of the attempt until its completion a tremor appears that involves the entire limb, and increases in amplitude as the act nears its completion. This is called an intention tremor, and is so definitely characteristic of the disease as to be almost pathognomonic. Early in the disease the tremor may not be associated with weakness;

later, however, weakness and rigidity occur, which are generally more marked in the legs than in the arms.

On lateral or upward movements of the eyeballs nystagmus appears early; later in the disease it may be constantly present. The pupils are often unequal, but always respond to light. Occasionally weakness or paralysis of one or more of the ocular muscles occurs. In many cases the optic nerve becomes affected, and frequently this affection of the optic nerve is an early sign of the disease. There is usually pallor of the optic disc, and atrophy limited to a portion of the optic nerve is very common. The visual disturbances in this disease may vary from day to day, and at times may even disappear.

The speech is frequently disturbed: it becomes very slow, the words being pronounced syllable by syllable, with pauses between; this condition is known as "scanning speech," and may be preceded for a considerable time by mere slowing of the speech. Apoplectiform and epileptiform seizures are uncommon. Attacks of uncontrollable laughter, of depression, of emotional disturbances, etc., occur without cause early in the disease. Affections of sensibility are rarely absent throughout the disease, but are seldom permanent or severe. The patient frequently complains of paresthesia and pains in the extremities, trunk, and head; these usually come on in attacks, and may be very severe. Disturbances of the rectal and vesical reflexes may occur, but complete or permanent paralysis is exceptional. In some cases there is a disturbance of the intellectual functions, a lack of interest develops, together with forgetfulness and confusion, but marked mental disturbances are rare.

When the spinal cord is first and principally affected, a spastic weakness in the lower extremities usually develops, which may or may not be accompanied by atrophy of the muscles, bowel and bladder disturbances, etc. In these cases bulbar and cerebral symptoms, by later development, reveal the true disease. In other cases bulbar symptoms are the first to appear, whereas in still others the cerebral symptoms may be the first evidence of the disease.

Diagnosis.—Multiple sclerosis is differentiated from *hysteria* by the fact that nystagmus, optic atrophy, the loss of control

of the sphincters, and the peculiar movements of the body and head do not occur in hysteria. A study of the case generally enables one to make the differentiation, but there are some cases in which there is an absence of objective symptoms which renders the diagnosis extremely difficult. From *lateral sclerosis* this disease is differentiated by the appearance of cerebral symptoms, such as scanning speech, nystagmus, and emotional disturbances, and by the occurrence of remissions, which are absent in lateral sclerosis. From *locomotor ataxia* it is differentiated by the presence of speech disturbance, nystagmus, and intention tremor. If multiple sclerosis begins with cerebral symptoms, it may be mistaken for cerebral tumor, vascular disturbances, or encephalitis. From *tumor of the brain* it is distinguished by the absence of symptoms of increased intracranial pressure and by the frequent presence of choked disc in tumors. Moreover, in tumors the symptoms usually increase gradually, whereas in multiple sclerosis there may be remissions and exacerbations. From *vascular disturbances* the differentiation cannot always be made if multiple sclerosis begins with an apoplectiform seizure, and only the further course of the disease will definitely determine the diagnosis. From *cerebrospinal syphilis* multiple sclerosis is differentiated by examination of the blood and spinal fluid.

Course and Prognosis.—The disease almost always runs a chronic course, with frequent remissions and sometimes even intermissions. In many cases there are repeated intermissions that may even simulate recovery; these intermissions may last for months, for a year, or even longer, and be followed by a reappearance of the symptoms, which increase in number and in severity, until, after a number of attacks, they become permanent and the disease progresses. The recurrences may be spontaneous, or they may be brought about by some harmful condition, such as exposure, overexertion, or following infection. The prognosis as to recovery is unfavorable, although some cures have been reported. The earlier this disease develops in life, the better would seem to be the chance of arrest. The appearance of bulbar symptoms should always give rise to apprehension, as there is great danger to life from respiratory involve-

ment and from disturbance of nutrition secondary to interference with deglutition.

Treatment.—There is no specific remedy for this disease. Mild hydrotherapy, gentle massage, and proper attention to nutrition and to elimination are all of service in staying the progress of the disease. Rest, both mental and physical, all movements being absolutely avoided, seems to be of the greatest assistance in arresting the progress of the disease.

CAISSON DISEASE

Etiology.—This is a condition which affects those individuals who work under increased atmospheric pressure, such as bridge-builders, divers, miners, etc., who, after being subjected to a pressure of from two to six atmospheres for some time, suddenly, without taking proper precautions, emerge from the high atmospheric pressure into one much lower.

Healthy men are much less likely to be affected by this disease than are those who overindulge in alcoholic drinks or who have heart or kidney diseases.

Pathology.—As a result of sudden decrease of atmospheric pressure, gas-bubbles escape from the blood, causing air emboli in the small vessels. In those cases that do not recover secondary softening of the cord or brain takes place. The extent of the softening depends upon the size of the vessel that is occluded. Fortunately, in the majority of cases the emboli are of microscopic size and are located in the capillaries, hence the areas of infarction are minute, and the collateral circulation is sufficient to bring about rapid restoration of the nutrition and function of the affected area. In those cases in which the larger vessels are occluded, softening of the cord or brain follows, which causes either chronic symptoms or death. In those cases that survive for a few days, air emboli are not found, but the areas of softening, with their degenerative changes, are seen.

Symptoms.—The symptoms develop within a very short time after the individual emerges suddenly from under increased atmospheric pressure to one much lower. The symptoms are both cerebral and spinal: there is a feeling of pressure on the

head, vertigo, tinnitus, vomiting, cramp-like pain in the back and legs, weakness or paralysis, and occasionally respiratory and cardiac disturbances. Some patients lose consciousness. In some cases the symptoms point almost entirely to involvement of the spinal cord: there occur pain in the back and legs, weakness or paralysis of the legs, sensory disturbances, and loss of bowel and bladder control. All these symptoms usually develop within a few hours. In some cases the symptoms subside almost as rapidly as they develop, and in a short time the patient has entirely recovered; in other cases the spinal or cerebral symptoms persist, and the symptoms either of myelomalacia or of cerebral softening develop.

Prognosis.—The prognosis is fairly good, except in alcoholics, and in those suffering from heart and kidney disease; even in these the prognosis is not necessarily bad if the condition is recognized and the individual returns rapidly to a chamber in which the atmospheric pressure can be increased.

Treatment.—It is most important to warn individuals against rapid changes from high to low pressure—the changes should be made very gradually. When the symptoms develop, the patient should be immediately placed under increased atmospheric pressure, either by returning him to the place from which he came, or by placing him in a pneumatic cabinet, where a high atmospheric pressure can be quickly produced, and then gradually returning him to the lower atmospheric pressure. Ergot in large doses every two hours and bandging of the limbs have been recommended, in order to confine the blood to the body cavities. After the acute stage of the attack has passed, the treatment is that usually given for spinal or cerebral softening.

DISEASES OF THE NERVOUS SYSTEM WITHOUT PATHOLOGIC FINDINGS

HYSTERIA

Hysteria is a condition characterized by the presence of an abnormal mental condition that manifests itself by an increased suggestibility, great emotional mobility, and the occurrence of symptoms of varying degrees of severity, involving the emotions, the motor, the sensory, and the vasomotor system, and frequently the occurrence of abnormal states of consciousness and of the mind.

Etiology.—This disease manifests itself for the first time most frequently between the age of puberty and the thirtieth year. It may occur earlier in life, being very uncommon bebefore the tenth year, although it may occur in very marked form in the earliest years of life. After the twenty-fifth year there is a decrease in the frequency with which the disease manifests itself for the first time. Women are affected somewhat more frequently than men. The disease is more common in the Latin, Hebrew, and the Slavonic races than in the Anglo-Saxon and the Teuton.

Heredity is a most important predisposing factor. Frequently there is a history of hysteria in the mother or father, and in other cases neuropathic taints, exhausting disease, alcoholism, etc., are discovered in the ancestry. Prolonged illness, overwork, worry, changes in one's surroundings, occupations that involve intense mental strain or anxiety, etc., may also act as predisposing causes. Among the exciting causes are fright, shock, worry, unhappiness, disappointment, fear, incompetence, inability to attain one's desires, imitation, homesickness, the adminstration of anesthetics, acute infectious diseases, toxemia, etc., or any combination of these causes may act as the direct excitant of a hysteric condition.

Theoretic Etiology of Hysteria.—Practically all authorities agree that in hysteria there is an underlying abnormal mental state that is of congenital origin, and that this mental condition is characterized by a more or less marked emotional mobility and susceptibility to suggestion. These individuals are usually rather superficial in their judgment, lack self-control, and are more or less egotistic and inconsistent in character. There is an exaggerated susceptibility to external or internal impressions, and, in addition, hysteric individuals are usually fearful, evasive, and unwilling to meet the disappointments of life in a frank manner. The hysteric tendency may remain latent and never become manifest. In many instances, however, as the result of disagreeable or unfortunate experiences occurring in early life, before puberty, during adolescence, and sometimes later, there is produced a form of mental distress which the individual is unwilling to face openly, and consequently the thoughts or the occurrences that give origin to this discomfort are repressed and disappear from consciousness. These repressed ideas still remain active, and become converted into some form of physical or mental reaction, which may evidence itself symptomatically in the shape of motor, sensory, vasomotor, or mental phenomena.

Many persons possessing a hysteric temperament show symptoms that are defensive reactions; such individuals, as the result of incompetence, unhappiness, a desire for attention, unwillingness to face their failures, disappointments, or neglect, *unconsciously* endeavor to cover over these deficiencies by symptoms of illness. In some cases, as the result of prolonged illness, overwork, insufficient food, worry, or toxic conditions, there occurs a more or less marked decrease in the normal powers of inhibition. In these individuals, in the presence of some acute, exciting cause, attacks occur that are characterized by symptoms of lack of self-control, such as immoderate weeping, laughing, trembling, restlessness, etc. It will also be observed that in these patients the emotions exert an abnormal influence upon the motor, sensory, and secretory functions. As a result of the exaggerated suggestibility, the expected or preconceived ideas are followed by exaggerated reactions of a negative or

positive kind, which may be of motor, sensory, vasomotor, or psychic character. For example, as the result of a fright, with or without actual injury, the individual may develop a paralysis or contracture, anesthesia, or great pain, etc.

Symptoms.—This disease is characterized by a more or less well-marked mental peculiarity. While this peculiarity is usually present, in some individuals it may remain latent and evidence itself only under unfavorable conditions. The hysteric state manifests itself in many ways: some individuals are timid and fearful, whereas others are bold and aggressive; some are changeable and inconsistent, whereas others are stubborn; some are generous, others selfish—but all show superficial judgment, are very susceptible to external or internal impressions, are abnormally suggestible, strongly emotional, and lack ability to exercise control over their emotions and actions. In some cases they seem devoid of the power of arresting some of the reflex bodily functions. These individuals are usually egotistic, crave sympathy, and are often of an introspective temperament, showing a marked tendency to exaggerate their symptoms.

Some hysteric persons who are conscious of a tendency to emotional outbreaks may succeed in controlling the attacks; in other individuals the hysteric temperament may not manifest itself. In both of these groups of cases any untoward condition, such as prolonged exhausting illness, overindulgence in alcohol, shock, severe fright, great grief or sudden joy, may precipitate an attack or reveal the presence of a hysteric temperament.

Those who are afflicted with this peculiar mental temperament are subject to attacks that may be of short or of long duration, and that may be mild or severe. Such attacks are characterized by disturbances of the emotions and of the motor, sensory, vasomotor, secretory, or mental functions. Generally more than one of these functions are affected, although it is quite possible for the attack to be characterized by either an increase or a decrease of the normal functions of any one of these elements. According to their severity and duration the attacks are described as minor or major. Either with or without the major or minor attacks, or following or preceding an attack, there may

occur evidence of distrubance of the bodily or mental functions, which may last a long or a short time.

As the result of an emotional or physical disturbance an attack may develop that lasts for a short time, and that is not characterized by marked loss of self-control. In these minor attacks the individual is usually restless, may be apprehensive, feel depressed, has a choking sensation, a peculiar feeling in the throat as if a lump were there which can neither be swallowed nor brought up (the globus hystericus). He may complain of a sense of weight on top of the head, pains in the eyes or back, or a pain as if a sharp object were being driven into the top of the head. There may be a sense of extreme weakness. Some patients are seized with an imperative desire to cry or to laugh, and in some attacks of laughter are followed by spells of weeping; in others the restlessness increases and the mental state simulates delirium. The attacks may cease suddenly without apparent reason, or the patient may fall asleep and awaken in a normal state, or under treatment the attacks may subside gradually. On the other hand, the patient may have a number of these mild attacks, one following the other at short intervals.

After recovering from a series of attacks or from a severe attack the patient may be considerably exhausted. In some individuals these minor attacks are characterized by the occurrence of severe pain, which may be localized or diffuse, may last a variable length of time, and then gradually subside. The pain is most frequently located in the head and the back of the neck.

The major attacks may begin with restlessness, emotional disturbances, a tendency to weep or to laugh, the presence of the globus hystericus, etc. In some cases the symptoms may become more marked, and the patient may scream, become more restless, eventually either running wildly about or falling upon the ground or upon some convenient place, and exhibiting violent muscular movements or becoming rigid. The convulsive movements exhibited by these patients are always extensive and purposive in character: they kick, bite, strike at bystanders, scratch, run their fingers through their hair, being careful, how-

ever, not to pull out their own hair. They suck their gums and lips to make them bleed and sometimes expectorate bloody saliva. Their contortions and movements may become most violent. Occasionally the individual becomes perfectly rigid, ceases to breathe for a few seconds, the face becoming cyanotic; in most instances, however, the face is flushed, and the skin is moist or covered with perspiration. Not infrequently during these seizures the patient assumes peculiar attitudes—opisthotonos; in more marked cases the arc de circle and attitudes expressive of some emotion are assumed.

During the attack the patient frequently screams, sings, laughs, and at times uses very profane and obscene language. Some patients utter meaningless sounds, as if they were speaking in a foreign language. There may be a perversion of consciousness, but complete unconsciousness practically never obtains, as these individuals usually react to external impressions. The attacks last for variable lengths of time; in some they are of only a half-hour's duration, or even less, and in others the attack may last many days. In some the attacks occur only at infrequent intervals, and then apparently follow some exciting cause; in others the attacks occur frequently without any definite causative condition. The attacks may be preceded by premonitory symptoms, but in these individuals the warning is usually variable, and generally occurs quite a while before the attack. As a rule, these attacks subside gradually and are followed by a period of marked exhaustion and gradual recovery.

In both the major and the minor attacks the pupils are usually equally dilated and react to light; the deep reflexes are generally increased, and the Babinski sign is absent. During the attack the individual does not injure himself, except accidentally; for example, in his struggles with those who seek to restrain him he may be injured, or he may accidentally injure himself by striking against some object. Following the attack he usually passes a large quantity of very pale urine. Instead of gradually recovering after an attack, some individuals may enter a state of delirium that may last for several days, and then gradually subside, when recovery sets in.

The hysteric attacks may vary greatly in character; in some, the attack is limited to loss of emotional control, at times accompanied by sobbing, uncontrollable weeping, or alternate laughing and weeping; or there may be restlessness, displays of temper, choking sensations in the throat, etc.; or anxiety, fear of death, pallor, weakness, profuse perspiration, tremor, and pain, or vertigo, dimness of vision, ringing in the ears, a feeling of weakness, may be the symptoms that constitute the attack.

Hysteric attacks may closely simulate major epileptic attacks, or they may be characterized by peculiarities of movements during which the patient acts as if he were performing some definite act, such as bowing or skipping, or he may suddenly assume an attitude or an expression and maintain that for a longer or a shorter period.

Attacks that closely resemble fainting spells may occur; usually tremor is present, either in the hands or in the face; the pulse remains unaffected.

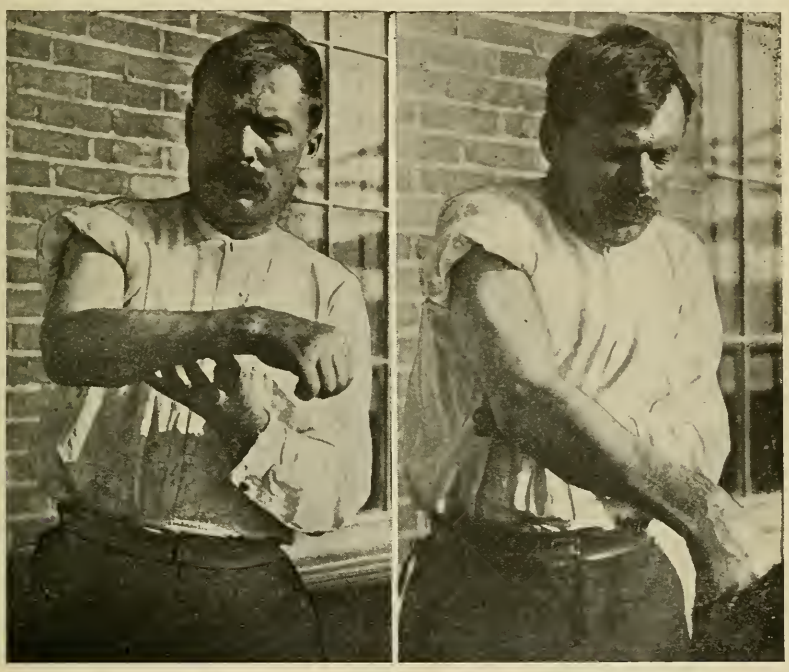
Attacks of sleep may follow an emotional disturbance or several of the ordinary hysteric seizures. The onset of these attacks may be sudden or gradual, and they may last from a very short time to hours, days, or months, terminating suddenly in a convulsive attack or by gradual stages. During this state the patient appears as if he were sleeping naturally. Although these patients are apparently unconscious, they are generally able to tell what has taken place during this period. The pulse-rate may be somewhat slowed; the pupils react to light; the respiration is generally somewhat slower. In this state the individual may be completely relaxed, but there is a tendency to fall into a cataleptic condition.

Somnambulistic Attacks.—After some emotional disturbance or hysteric attack the patient suddenly acts as if he were affected with some abnormal mental state. He may remain in this condition for a considerable length of time, during which he seems possessed of another character and acts in accordance with the same. While in this state his physical actions correspond to his newly acquired character. When the spell has passed off, the individual not infrequently finds himself amid new sur-

roundings, and has no recollection of the events that have taken place during the attack. In succeeding attacks he may recall everything that transpired during previous seizures.

Attacks of this sort frequently give rise to conditions that are known as double personality.

Following a major or a minor hysteric attack, a terrifying experience, shock accompanied by emotional disturbance, fear, anxiety, disappointment, etc., there may be disturbances of



A *B*

Fig. 152.—*A*, Hysteric paralysis; *B*, making effort to raise arm.

motion and sensation, and of the vasomotor, secretory, or mental functions. These have no anatomic basis, and are the result of a disturbed activity, either increased or decreased, of the subconscious cerebral centers. These abnormalities of functions are in some instances capable of being reproduced voluntarily, but more frequently they cannot be voluntarily reproduced nor sustained to the extent to which they occur.

Disturbance of the Motor Functions.—Paralysis or contrac-

tures, or paralysis and contractures (Fig. 152), are of frequent occurrence. The paralysis may affect the entire limb, giving rise to a monoplegia, or it may affect the entire side of the body, causing a hemiplegia. It may affect both lower extremities, causing a paraplegia, or may involve all four extremities. In some cases the paralysis is limited to only a segment of the limb, usually involving the peripheral portion of the limb, although at other times the proximal part of the limb is affected. Instead of a complete inability to move all or a part of the body, in some cases a weakness exists. Not infrequently automatic and customary movements are carried out with the normal strength, but when such patients are requested to perform some voluntary movement, a weakness is found to exist. Examination reveals the fact that although all the muscles are well contracted, little or no effort is exerted. When a complete paralysis exists, the individual states that he is unable to move the affected parts, and when requested he makes no effort to do so. The paralysis in these cases is usually of the flaccid type, but the muscle tone remains normal and the deep reflexes are usually increased. The individual often unconsciously moves the part of the body in which he asserts that paralysis exists. The paralysis or weakness is generally limited to the extremities.

In some cases, instead of a paralysis or a weakness being present, the muscular movements are retarded. Voluntary movements carried out as the result of commands are performed slowly, and after a distinct interval, as if the patient had difficulty in associating the idea of movement with motor activities.

In other patients, instead of a loss or decrease of motion, abnormalities of movement occur. These may take the form of a contracture, under which condition an extremity, part of an extremity, the trunk, or the facial muscles are held constantly in a rigid position.

Contractures result in positions of the affected portion of the body that can be assumed voluntarily, and are further characterized by the fact that they are usually increased on attempts to overcome them, and likewise by the fact that they disappear during sleep.

Tremors and Other Abnormal Movements.—Tremors are usually coarse, but may be slow or rapid. They are generally increased upon voluntary movement and during excitement. As a rule, they are jerky in character, and often disappear when the patient is at rest or does not know that he is being observed. Grosser abnormalities of movement may also occur and may be choreiform in character, or movements may occur involving the whole body; thus the patient may go through swimming movements, dancing movements, sucking movements, etc.

Astasia-abasia is a peculiar motor condition seen to occur in hysteric individuals. In this condition the individual can, while sitting or lying, perform movements with his limbs with a normal amount of power, but is unable to stand or to walk. When the patient is helped to his feet, he may either collapse, or, after making one or two efforts to walk, he sways from side to side and staggers and falls.

Among the commoner forms of abnormalities of movement are: Singultus, which is repeated swallowing, generally followed by eructation and rumbling noises in the abdomen; the eructations and rumbling in the intestines are the result of the swallowing of air; hiccoughing; yawning; sneezing, which may be repeated for hours at a time; abnormalities of respiration in which the breathing is rapid and irregular; somewhat less common are hysteric blepharospasm and hysteric coughing, the latter being hoarse, loud, and frequently repeated. At other times the patient utters peculiar noises, and there is an inability to swallow; this may be due to a hysteric spasm of the pharynx and the esophagus. Hysteric aphonia is a condition in which the patient more or less suddenly loses his voice, being unable to speak above a whisper, and in some cases being unable to utter a sound.

Sensation, like motion, may be lost, increased, or perverted. One of the commonest complaints in hysteria is of pain, which may be said to be constantly present, or to occur in paroxysms and involve any portion of the body. Headache is very frequent. These individuals often complain of sensitiveness of the scalp; of very acute pains in circumscribed areas of the head

and of occipital headache; pain radiating through the head into the eyes; drawing pain in the back of the head; headache limited to one-half of the head, etc. Pain of a neuralgic character; pain in the back; pain limited to one-half of the body; pain in the breast (mastodynia); pain described as existing in various organs; pain in the extremities; pain in the lower end of the spine; pain in the joints; pain simulating angina pectoris, etc., are also frequently complained of. These painful states are characterized by the fact that careful examination does not reveal any organic cause for their presence. They may be very persistent, lasting for weeks and months. They seem to have little effect upon the patient's general condition, and are markedly influenced by the mental state of the patient. Sometimes hyperesthesia of the skin and vasomotor disturbances occur in these painful areas.

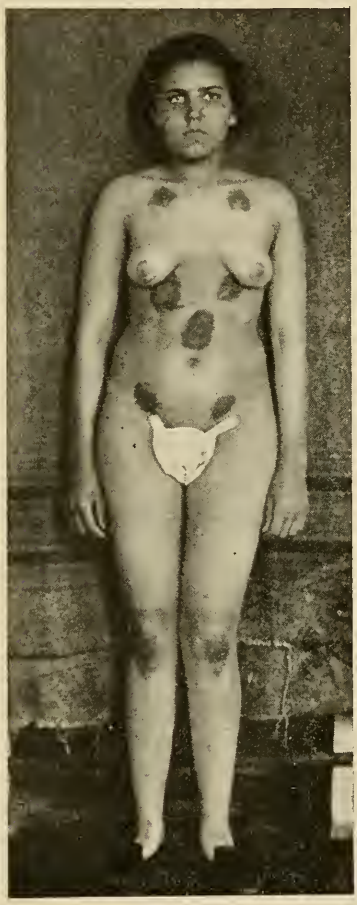


Fig. 153.—Hysterogenic zones.

Paresthesias varying in type are also frequently complained of in hysteria, as are also hyperesthesia, numbness, tingling, formication, etc. The distribution of these paresthesias may vary, like the distribution of pain from a localized area of one portion of the body to a more or less generalized distribution.

Hysterogenic Zones.—These are circumscribed areas that are usually hyperesthetic, and give rise to pain when pressed upon. These zones may be situated in different parts of the head, in the axillary line, in the submammary region, subclavicular region, over the sternum, in the lateral hypogastric region,

especially over the ovaries, in the epigastric region, and over the site of the apex-beat of the heart (Fig. 153).

The special senses may also be hyperesthetic, the patient complaining of extreme sensitiveness to light, to sound, to smell, etc., or there may be discomfort in using the eyes. This discomfort may be experienced only under certain conditions, such as on reading or upon looking at near objects or at colored objects, etc. Some patients complain of auditory sensitiveness only under certain conditions, some sounds causing great discomfort, whereas other pass unnoticed.

Decrease or loss of sensation is also common in hysteria. The distribution of the anesthesia is never confined to areas supplied by nerves or nerve plexuses or by spinal-cord segments: it frequently affects half of the body, being sharply marked off in the middle line, and sometimes involving the entire body. It may be limited to one arm or to one hand, the head or the face, to one leg or to one foot, both feet, etc. It is limited to areas of the body other than those observed in organic diseases, the areas of anesthesia being usually sharply defined. As a rule, they can be transferred from one side of the body to the other, and be made to appear and to disappear under appropriate suggestion. The mucous membranes may also be affected. When sensation is involved, pain, tactile, and temperature senses are all usually affected; the degree of involvement may vary from slight reduction of sensibility to complete anesthesia. In some cases there may be dissociation of sensation, there being loss of the sense of pain without impairment of the sense of touch (Fig. 154).

The special senses may also show a similar change. Among the most common and important are the visual disturbances. In some cases there is a diminution in the acuteness of vision, with, at times, a concentric narrowing of the visual field. Rarely a bilateral hysteric blindness is present. In these individuals the pupillary reflex is retained, and the individuals find their way about with comparative ease, avoiding obstacles. Very frequently a hysteric ptosis is combined with the hysteric amaurosis. Decrease or loss of the sense of smell, sense of taste, and sense of hearing may also occasionally occur. Hysteric pa-

tients are frequently ignorant of the sensory disturbances, and are made conscious of them only at the time of the examination.

Reflexes.—The superficial reflexes may be decreased or abolished or remain unaffected. The tendon reflexes are usually

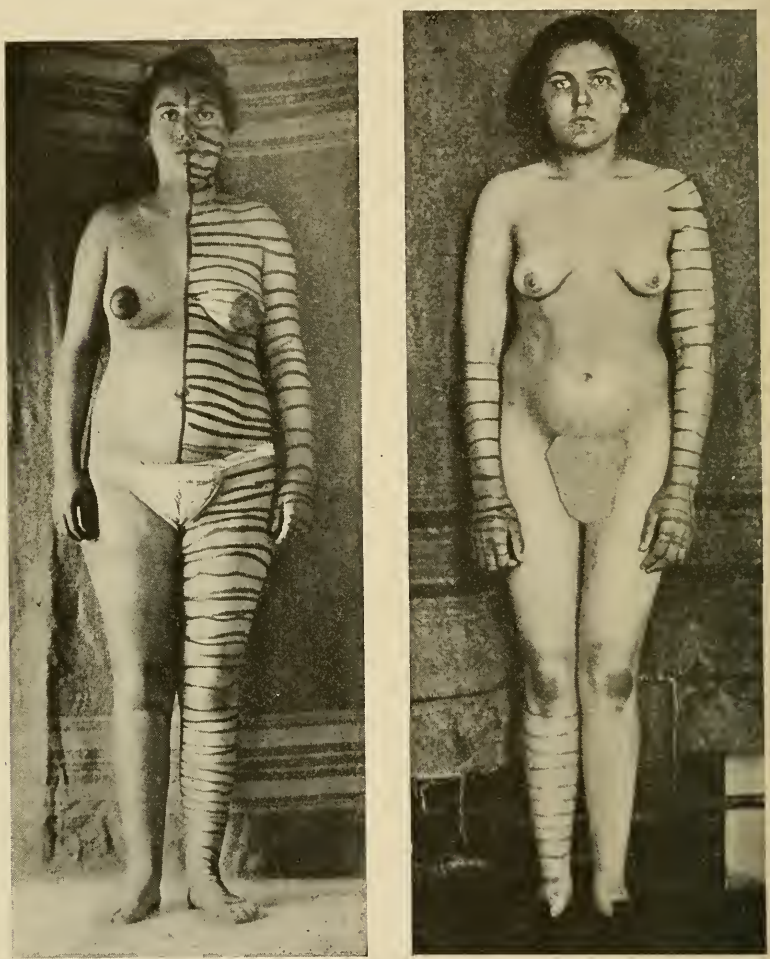


Fig. 154.—Distribution of anesthesia in hysteria.

increased. The pharyngeal and palpebral reflexes disappear if there is decreased sensation in these parts. The cardiac and respiratory reflexes to painful cutaneous stimuli are retained, even when an anesthetic zone is irritated.

Vasomotor and circulatory disturbances frequently occur; in patients with hysteric anesthesia often a needle-prick does not cause bleeding, and sometimes operations upon the superficial tissues can be performed with little loss of blood. Paroxysms of cardiac palpitation, with rapid pulse, rapid changes in color, sudden pallor, and flushing commonly occur. Hard, inelastic, circumscribed swellings may appear that do not pit upon pressure, and that may be pale or bluish in color.

Disturbance of the Secretions.—There may be a marked increase or decrease in the amount of saliva secreted, and the amount of urine voided may be greatly increased. As a rule, large quantities of urine are voided after an attack.

Gastro-intestinal Disorders.—Some patients complain of complete loss of appetite and a distaste for all kinds of food. These symptoms may be present only during attacks or may be persistent. Occasionally there is marked increase in the appetite, and in some individuals loss of appetite alternates with marked increase of appetite. If a loss of appetite with refusal of food persists for any length of time the patient's nutrition becomes affected and the condition is attended with a certain amount of danger.

Hysteric vomiting is a somewhat common symptom. The food is generally vomited immediately after it is taken. As a rule, the appetite remains good, and the individual does not become emaciated. This symptom may be very persistent.

Protrusion of the abdomen, caused by distention of the intestines with gas and by a relaxed condition of the abdominal muscles, and loud rumbling noises in the intestines are frequently present in hysteric individuals. Constipation may continue for days, or diarrheal attacks may be present.

Trophic disorders are certainly uncommon; they may occur as the result of prolonged pressure in hysteric contractures, but are usually of traumatic origin, and are sometimes produced voluntarily.

Hysteric fever is not a common symptom, and when it does occur the greatest care should be taken to exclude organic disease. The fever may be continuous or intermittent in type, the temperature ranging from 100° to 111° F. These febrile

attacks are usually unaccompanied by alterations in the pulse and respiration.

In my experience the rise in temperature has not been accompanied by an increased warmth of the skin or mucous membranes; but was, rather, the result of manipulation of the thermometer on the part of the patient. One must, however, admit the possibility of the occurrence of a true hysteric fever.

Mental Symptoms.—The mental faculties are generally unimpaired. Hysteria occurs more frequently in intelligent and sensitive individuals than in the stupid and phlegmatic. Hysteric individuals are usually very impressionable, their thoughts being more or less self-centered, and directed only toward matters in which they are interested; this frequently gives rise to an inattention to other matters, with corresponding neglect. The memory, as a rule, does not show any marked impairment, but as the result of inattention it apparently may be weakened.

Some individuals find certain subjects disagreeable, and ideas may be fixed in the memory with an increased tenacity, and be more or less persistently present. In others there may be an apparent loss of memory for special events or times. These disturbances of memory may occur periodically, and during these periods the patient may remember the events that occurred at a preceding period, but during the intervals is entirely amnesic for events and occurrences that have taken place during the attack.

These memory defects are especially apt to follow repeated hysteric attacks.

Catalepsy is a condition that is characterized by rigidity of the extremities. This rigidity may be general or limited, and may result in the patient remaining in the position in which he is placed for considerable lengths of time. Attempts to move the limbs are met with slight resistance; the sensibilities are usually decreased or abolished; the individual is not unconscious, but is generally in a dreamy state.

The pupillary reflexes are always retained. The patient may remain in this state for hours, days, or weeks. This condition usually follows some mental excitement or may occur after a hysteric attack.

It is important to differentiate this condition from dementia præcox; this can be done only after a very careful consideration of the history and on prolonged observation.

Course.—In the great majority of cases the disease runs a chronic course and is subject to many variations. Generally intervals occur during which the individual is apparently normal. The character of the attack may vary from time to time, and there may be complete disappearance of some of the symptoms with the appearance of others. In certain cases, particularly those occurring during childhood, there is a predominance of some special symptoms, and sometimes only one symptom may be present.

The disease may develop gradually or the onset may be acute, beginning with subjective symptoms, to which are added, from time to time, objective manifestations, which appear particularly after sudden emotional disturbances or injury. The disease manifests itself most frequently for the first time between the twelfth and the twenty-fifth year, and generally following some form of emotional disturbance. Recurring attacks appear which vary in severity and in character, and occur at irregular intervals. They are usually associated with emotional disturbances, apprehension, or fear, either of external or of internal origin, and the symptoms vary. The condition may last for many years. In a large number of cases the symptoms become less severe and sometimes disappear after the fiftieth year.

Prognosis.—The prospects of recovery are better if the patient has confidence in the one who administers the treatment, and if there is an incentive and a determination to recover. The prognosis also depends on the possibility of removing the cause, or upon the ability of the patient to resign himself to existing conditions.

In those individuals in whom a marked hereditary tendency exists and who present stigmata of degeneration the probability of recovery is slight, whereas in those in whom there is an absence of a neuropathic hereditary tendency and in whom the hysteric symptoms appear only after some acute condition a very favorable prognosis may be given. When

hysteric symptoms are combined with great anxiety, hypochondriasis, neurasthenic states, or psychosis the outlook is extremely unfavorable.

Under proper treatment the vast majority of cases recover from the hysteric attack, and there may be total disappearance of the hysteric symptoms. In some cases, even under the most expert treatment, hysteric symptoms persist for many years. The prognosis as to recurrence of attacks or of symptoms is unfavorable, owing to the fact in these individuals a peculiar mental state exists that predisposes them to a recurrence of the condition. There are, however, many individuals who, under proper treatment, can be brought to realize their weakness, who are willing and anxious to avoid further attacks, and who finally succeed in overcoming this tendency and remain permanently free from the symptoms.

Diagnosis.—The diagnosis must be made by exclusion. Hysteria may resemble any of the organic physical or nervous diseases, and it is, therefore, necessary to make a most careful examination of the internal organs and of the nervous system.

In differentiating hysteric from organic diseases it is extremely important to examine the fundus of the eye and to detect any pupillary disturbances; the distribution and character of paralyses and of sensory disturbances; the presence or absence of the deep and the superficial reflexes; the presence or absence of bowel and bladder disturbances; the nature of speech disturbances; the character, duration, and sequelæ of convulsive attacks; the reaction of symptoms to external influences, etc., as guides to a correct diagnosis.

If a proper examination has been made, error is possible only under the most unusual conditions. It is important to differentiate hysteria from such diseases as neurasthenia, epilepsy, edementia præcox, and from conditions whose principal symptom is pain, as, for example, neuralgia, headache, painful joints, angina pectoris, etc.

In differentiating hysteria from neurasthenia the following points are important:

	Hysteria.	Neurasthenia.
Onset:	Generally sudden.	Gradual, unless traumatic.
Course:	Changeable, irregular, and paroxysmal.	More or less monotonous.
Seizure:	Convulsive attacks, crying or laughing spells, etc.	Absent.
Effect of effort:	Frequently dispelled or decreased.	Aggravated or rendered worse.
Intervals:	Of comparatively normal capacity.	Uniform incapacity.
Sensory disturbances:	Definite areas of anesthesia.	Absent.
Painful areas:	Hysterogenic zones sensitive.	General sensitiveness.
Contractures:	Frequently present.	Absent.
Motor disturbances:	Paralysis, gross abnormality of movements, choking sensation, etc., often present.	Complaint of general weakness.
Visual fields:	Frequently contracted.	Undisturbed.
Mental state:	Exaggerated suggestibility.	Pathologic fatigue.
Emotional state:	Emotional mobility, with defective emotional inhibition.	Uniform emotional depression.

In neurasthenia there is a history either of decreased mental and physical power, dating back to earliest childhood, or of overwork under unfavorable conditions. In hysteria there is a history of lack of emotional control, of egotism, of peculiar attacks, and of pathologic suggestibility during childhood or early adult life, or of some emotional shock, disappointment, fear, failure, etc., preceding the illness.

The differentiation between epilepsy and hysteric attacks is discussed under Epilepsy.

In distinguishing hysteric painful seizures from such conditions as neuralgia, examination reveals the fact that in hysteria the pain is rarely confined to the area supplied by a nerve; in hysteria nerve pressure-points are, as a rule, not so sensitive as in neuralgia; in hysteria tenderness is diffuse, whereas in neuralgia it is confined to nerve pressure-points or to the area of distribution of the affected nerve. In hysteria the pain can be influenced by mental suggestion and the attacks are usually dependent upon some form of excitement.

In distinguishing the headache of hysteric attacks from a true

headache a careful examination is always required: in hysteria the pain is apt to be limited to circumscribed areas, and is, as a rule, dependent upon some form of emotional disturbance; it comes on more or less suddenly, and frequently disappears with equal suddenness; prolonged pain does not affect the general physical condition, as does the prolonged and severe pain that is the result of organic disease.

The differentiation between hysteric painful joints and hysteric pains about the heart and pain in the joints as the result of organic disease and of organic cardiac affections attended by pain can usually be made if one takes into consideration the absence of physical findings and the history of a preceding emotional disturbance in the former case, and the presence of physical findings and the history of some preceding disease and emotional disturbance occurring after the pain in the latter case. The differentiation between the early manifestations of dementia præcox and hysteria is in some cases extremely difficult. In dementia præcox there is a history of a previous normal mental condition or a peculiarity of mental temperament: in hysteria there is a history of emotional hysteric temperament; in dementia præcox the actions are somewhat purposeless, whereas in hysteria they seem to follow a definite purpose—that of impressing those with whom the individual comes in contact.

In dementia præcox there are more or less indifference, marked irritability, and frequently ideas of reference; in hysteria the individual craves sympathy, anger alternating with affection, and the individual blames his condition on an unfortunate combination of circumstances, but not on others.

Treatment.—Persons in whom there is a history of a hereditary tendency to nervous disease or who show evidence, early in life, of a hysteric temperament, should have their lives so ordered that they are assured, as far as possible, of a normal physical development. Their education should be so directed that they may acquire altruistic tendencies, and they should be taught the value of self-control.

In the early years of life much can be done for these children: if the mother is hysteric or nervous, she may set a very bad

example for the child by speaking of her own illness or by complaining in the presence of the child. Undue importance must never be given to the child's complaints. Every effort should be made to teach it self-control. The child's attention and interest should be directed toward external objects; thus it should be encouraged to observe the phenomena of nature and be interested in outdoor sports. The child should be protected from excitement, and the reading should be carefully supervised. It is often advisable to separate the child from the home conditions and place it amid new surroundings. Careful physical examination should be made, and all physical defects or illnesses should receive proper treatment. The diet should be regulated, and should consist of simple, easily digested, and nourishing food. Alcohol should be strictly interdicted.

The occupation of an individual with a hysteric temperament should be carefully selected, and should be one as free as possible from worry, strain, and emotional excitement.

The treatment of the hysteric state that has already developed is at times very simple, and at other times extremely difficult.

Before undertaking the treatment of this condition it is necessary to make a thorough and careful examination. This is done for two reasons: To discover the presence of any physical or mental abnormality that may require treatment, and to gain the patient's confidence; the latter is a most important aid in carrying out the treatment. It is usually necessary to remove these individuals from their accustomed environment, and in the more serious cases complete isolation from family and friends is absolutely necessary.

The Treatment of Hysteric Symptoms.—Having gained the patient's confidence, and having placed him amid favorable surroundings, it is important to direct the treatment to the relief of his symptoms. As these symptoms are entirely psychic, their removal is possible only by influencing the mind of the patient. He should be assured that the illness is not serious, and in many cases a frank explanation of the cause of the symptoms and an effort to arouse the coöperation of the patient are sufficient to bring about an improvement. There are many

patients, however, who, during the earlier stage of the treatment, are incapable of facing their illness frankly, or are unwilling to do so. Such individuals must be relieved of their symptoms and must gradually be brought to a mental state in which they will coöperate with the physician; this can be brought about with the help of drugs or of some physical measures, such as electricity, hot or cold packs, the cautery, etc. The nature of the drug or the physical means one employs is of no importance, provided its use is accompanied by the suggestion, on the part of the physician, and the hopeful attitude, on the part of the patient, that a favorable result may be expected. It is better to employ small doses of aromatic substances, such as tincture of valerian, tincture of asafetida, etc., or small doses of the bromids, frequently repeated, than larger doses at more infrequent intervals.

Treatment of Anesthesia.—This is often markedly benefited by the application of the faradic current through a metal brush, or by the spark electrode from a static apparatus. These applications must, of course, be accompanied by the suggestion or statement of the physician that they will be effective. At times the internal administration of drugs, when accompanied by the assurance that relief will follow, results favorably. Painful conditions, such as headache, backache, neuralgia, etc., may be relieved by inducing counterirritation, as by mustard foot-baths or the cautery, rubbing with liniments, either locally or at a distance from the seat of pain.

Static electricity, the violet-ray, massage, the galvanic current,—using the constant pole over the painful areas,—or even the faradic current may be of value when accompanied by proper suggestion.

In prescribing drugs for painful conditions it is best to give frequent repeated small doses. At first tincture of valerian, given in doses of 5 drops every two hours, or potassium or sodium bromid, 5 grains every two hours, may be prescribed. If these remedies fail, phenacetin or aspirin in small doses should be given. In many cases a placebo will be as effective as any of the aforenamed drugs. Morphin and chloral, and drugs of a similar nature, should always be avoided.

Difficulty in swallowing, or dysphagia, may be relieved by gargling, the passing of a stomach-tube, or the application of the faradic or the galvanic current to the neck. At times painful conditions are relieved by the giving of an emetic and the production of vomiting. Hysteric nausea, vomiting, eructations, etc., may be relieved by changing the diet, applying counterirritation over the stomach, and the application of the faradic or the galvanic current to the abdominal wall; sometimes it is necessary to apply the galvanic or the faradic current by means of an intragastric electrode.

If drugs are used, it is advisable to administer them in the smallest possible doses; thus the giving of 1 or 2 drops of almost any drug, if accompanied by the proper suggestion, is more apt to yield a favorable result than the administration of larger doses.

Paralytic conditions are best treated by persuasion, suggestion, and passive movements; if these fail, various physical means should be employed, such as hot and cold sponges, counterirritation, massage, and electricity.

It is most important to encourage these patients and to have them make efforts to use the affected parts as soon as possible. The treatment of contractures and abnormalities of movement is the same as the treatment of paralysis, but must, of course, be accompanied by different suggestions.

In treating hysteric aphonia or a hysteric cough, in addition to the measures enumerated above, it may be necessary to apply local treatment to the throat or larynx, always, of course, accompanied by suggestion.

Treatment of Hysteric Attacks.—The attacks can sometimes be avoided by applying pressure upon some particular part of the body. If an attack has set in, it is advisable to remove the patient to a quiet room, excluding every one but the person who is to have charge of the patient. Sometimes the application of cold water to the patient's face, the inhalation of spirits of ammonia, or the giving of a small dose of aromatic spirits of ammonia may terminate the attack. In other cases the administration of medicaments having a disagreeable taste, fre-

quently repeated, is efficient. It is most unwise to struggle with the patient or to attempt to restrain him.

Counterirritation by means of mustard-plasters or mustard foot-baths, or the application of the faradic current, also produces good results. Too much attention must not be paid to the patient, and the nurse must assume a strictly impersonal attitude and avoid all expressions of sympathy.

The after-treatment of the individual who has had a hysteric attack or who exhibits hysteric symptoms is just as important as the prophylactic treatment in the child.

It has been my experience that a large percentage of persons with a hysteric temperament, if properly treated, can be educated to exert emotional self-control, self-denial, and to acquire a spirit of contentment in their accustomed surroundings. In the large percentage it is advisable, and sometimes absolutely necessary, to remove them from their usual surroundings, and place them in a hospital or in some place where they will come in contact only with strangers. Here the physician will have complete control of the patient, and he will meet individuals who will set him a good example in the exercise of self-control. In the severer cases absolute isolation, excepting only physician and nurse, is necessary.

With the patient under these conditions, it is first necessary to correct any existing physical ailments. The diet should be regulated, and the patient's general physical condition brought to the normal. His powers of resistance are to be increased by the giving of cold and hot sponges, and his strength is to be augmented by proper exercise. The bowels are to be regulated by the administration of laxatives and by the promotion of regular habits. Any symptoms that may occur are to be met either by frank explanation and suggestion or by the use of medicine or physical means, accompanied by proper suggestion.

During the entire time that the patient is under treatment the intellectual intercourse between physician and patient must be of a stimulating, cheering nature. The patient is to be constantly reassured and encouraged, and every effort should be made to establish in the patient a determination to overcome his disability and to exercise control over his emotions—to

become, as far as possible, master of himself. He should be warned that failures and relapses are to be expected, but that with determination and constant struggle on his part he will eventually win the battle and become master of the situation. The future of these patients must be carefully directed for a considerable length of time. They should be encouraged to meet excitement with indifference, and to avoid, so far as possible, emotional disturbances and distressing incidents; to live a regular life, and to avoid mental and physical excesses. They should not give undue attention to minor physical ailments, but rather than remain anxious and uncertain it is advisable for them to consult a physician to discover the true nature of such ailments.

In the treatment of these patients it is of inestimable value to discover the cause of the symptoms.

Many patients, particularly if they have confidence in their physician, will, after careful inquiry, frankly unburden their minds and acknowledge the cause of the attack; there are, however, some few cases who either do not know the cause of their condition or are firmly convinced that no emotional disturbance has produced it; such patients find it practically impossible to unburden themselves, and consequently the unpleasant experiences are repressed into the subconscious mind and act as psychic irritants; such irritating processes must be dispelled or they become converted into physical manifestations and evidence themselves in hysteric symptoms and attacks. It is in this *very limited* class of individuals that much is to be expected from a psycho-analysis (performed by a properly trained physician) that tends to discover and to bring to light those psychic irritants. It is by freeing the mind of irritating experiences that one may relieve the patient's symptoms.

NEURASTHENIA, OR NERVOUS EXHAUSTION

Neurasthenia is a very common disease, occurring more frequently in individuals who live in large centers of population. Attention was first prominently called to this disease in 1869 by an American physician named Beard, who applied the name

neurasthenia to a state of chronic nervous fatigue and to the symptoms that were caused as the result of a chronic pathologic exhaustion of the nervous system. The disease had, of course, existed, been recognized, and described before this time. It would appear that under the stress and strain of modern civilization this disease is occurring with much greater frequency than formely.

Etiology.—Heredity is the most important predisposing factor. In many cases careful inquiry into the family history shows the presence of mild hereditary taints—of debilitating or toxic conditions in the parents preceding the conception and birth of the affected individual. In the type of neurasthenia that develops in young children and young adults after very minor exciting causes (neurasthenia præcox) there is generally a history of marked neuropathic taint in the ancestry. These individuals are born with a deficient stamina and weakened reserve force of the nervous system, which, with or without exciting cause, becomes easily fatigued, and regains a normal state only with difficulty, if at all.

Sex.—It occurs in both males and females, but with greater frequency in males.

Age.—The disease manifests itself most frequently between the twentieth and the fiftieth year—at the time of life when the individual is putting forth his greatest efforts, often under unfavorable conditions, to attain success. The disease may occur at any period, neurasthenia præcox appearing sometimes in the very earliest years of life. It may also appear after the fiftieth year.

Occupations that involve confinement, close attention to detail, anxiety, great concentration, responsibility, etc., predispose to this condition.

Neurasthenia occurs more frequently in persons of an excitable and emotional temperament than in those of a phlegmatic disposition. Any disease or condition that lowers the general health predisposes to nervous exhaustion. Under unfavorable conditions neursathenia may develop after any of the infectious diseases, particularly after influenza, following severe hemorrhages, diseases of the genital organs, eyes, nose, or throat.

It may occur in the wake of prolonged intoxications, whether of endogenous or exogenous origin. Syphilitic infection, by bringing about a decreased physical resistance, and owing to the fact that it is usually accompanied by marked emotional disturbance of a depressive nature, may be an etiologic factor, as may also uncongenial employment, sudden changes in one's habits, overwork, sexual excesses, and the like.

Overwork with insufficient rest, anxiety, worry, and trauma, in the presence of any of the predisposing factors, are the most frequent exciting causes of the disease.

The commonest and most important cause of neurasthenia is mental or physical overwork, with insufficient rest, carried on in an atmosphere of anxiety and worry. It is seen oftenest in individuals whose general health has become debilitated by injudicious or unhygienic living, or as the result of disease or intoxication, and in whom there may or may not be a tainted hereditary history.

Pathology.—A careful examination of the nervous system fails to reveal any organic changes. A consideration of the symptoms, course of the disease, and the results obtained by treatment, taken together with the examination of the central nervous system, leads one to the conclusion that in neurasthenia there exists a state of fatigue or exhaustion of the cells of the central nervous system; that these cells do not regain their normal state after the usual amount of rest, but that in many cases a comparatively normal state may be reëstablished as the result of prolonged rest combined with proper attention to nutrition.

If this conception of neurasthenia is true, the neurasthenic state differs only in degree from the fatigue which every one experiences as the result of overwork; consequently the amount of rest required in the neurasthenic state must greatly exceed the amount that would relieve normal fatigue. The mental state of the individual suffering from neurasthenia is also very important—these individuals are usually depressed, introspective, anxious, and apprehensive; mental rest is, therefore, very difficult to attain. As the unconscious mental activity is just as exhausting as, if not more so than, conscious and phys-

ical unrest, a vicious circle is established whereby the nervous system is maintained in a state of exhaustiou. It is the presence of this vicious circle that renders the establishment of a state of complete rest so difficult, and consequently makes the cure of this condition doubtful and sometimes impossible.

The mental state is caused by difficulty in the complex inter-associative activities of the higher cerebral centers. This difficulty in association and in interactivity is caused by the exhausted state of the nerve-cells. The mental condition is usually associated wirh emotional disturbances, and these in turn cause or are accompanied by abnormalities in the functions of the sympathetic system. These latter bring about abnormal states in the vasomotor system, the secretions, and the somatic sensibilities; sensations depending upon abnormal functioning of these structures are reflected upon consciousness, and in turn give rise to fear, anxiety, uncertainty, etc., thus tending to intensify the emotional state.

As a further result of the fatigue or exhausted state of the cerebral cells their power becomes diminished, and voluntary efforts are put forth only with difficulty. There is a feeling of deficiency, incompetency, and weakness, and as a result of these feelings impatience and irritability manifest themselves.

In many persons in whom there is a marked predisposition to the development of a neurasthenic state physical peculiarities are present, the most common of which are scoliosis, gastroptosis, enteroptosis, visceroptosis, floating kidney, flat, narrow chest, disturbances of vision and refractive errors, absence of muscle tone, etc.

Symptoms.—The symptoms of neurasthenia are practically innumerable. Every function of the body may become disturbed, and either be reflected upon consciousness, giving rise to discomfort, or manifest itself in some variation from the normal.

A careful examination of these disturbances of function will reveal the fact that while a deficiency is present, there is never a complete loss of the functions. Added to the deficiency there is usually an increased reaction to stimuli, generally of a disagreeable nature.

All the symptoms of neurasthenia are due to a state of fatigue

involving the various structures of the body; consequently, when voluntary effort is made to use these various structures, the response is brought forth only with effort, and the resulting activity is usually incomplete and occasions dissatisfaction in the mind of the individual, who feels his incapacity and inability to produce the desired results, and therefore becomes impatient, apprehensive, and irritable. Being in a state of fatigue and irritability, the patient becomes hypersensitive to many forms of stimulation, and the fatigue occasions discomfort which manifests itself in the form of pain.

Owing to the fatigue and irritable state of the mind, emotional disturbances have an increased influence on the mental activities, and are also frequently reflected, in an exaggerated manner, upon the physical functions of the body.

In neurasthenia a rapid fatigue manifests itself in the disturbed activity of practically all the functions of the body. In many cases disturbed activity of some organ or system of the body stands out prominently in the disease picture.

A predisposition to local symptoms is due either to a congenital weakness or to an exhausted state of an organ or system of the body as the result of overwork or of impaired general health by disease, combined with overwork of that portion of the body.

The *mental state* in this disease is one marked more or less by apprehension. The patient is irritable and introspective, and mental activity occasions a sense of discomfort. The individual complains of inability to fix his attention for any length of time upon any one line of thought, and there is a tendency for his thoughts to wander; he also complains of weakness of memory which, in fact, does not exist. This apparent weakness of memory is due to the inability of the patient to concentrate his attention upon one line of thought, and consequently the impressions are very superficial and soon disappear. This defect of memory applies only to unimportant happenings—events or matters of importance are well retained. There is inability to concentrate thought and to accomplish work that requires prolonged mental application. The patient can come to no conclusion, indecision being a prominent symptom.

Mental activity lacks vigor and is somewhat retarded; consequently these individuals complain of difficulty or inability to think. The intellectual powers are never greatly impaired. Fatigue prevents long-sustained mental application.

The emotional tone in these individuals is generally one of depression. Thus they weep, either openly or with some attempt at repression, when relating their symptoms or when contemplating their future. As a result of physical and mental weakness a sense of fear develops. Such persons, being introspective, misconstrue their symptoms and develop various phobias; these are always associated with a sense of discomfort or incapacity in some organ or system of the body; at times these fears become associated with some external condition, and may recur whenever the individual finds himself in a similar situation.

Among the commoner forms of fear is that of insanity—the patient notices that he has difficulty in remembering and in fixing his attention, and that there is inability to concentrate the thoughts, and that these symptoms are the forerunners of a mental breakdown. When he has attacks of palpitation, shortness of breath, or pain in the chest, he fears heart trouble or pulmonary disease; pain in the back makes him apprehensive of kidney or spinal trouble; weakness and pain in the legs make him fear locomotor ataxia, etc. Sometimes attacks of palpitation occur in crowded places or while the patient is alone or under some unusual condition, and subsequently he fears to place himself in a similar situation for fear of a recurrence of the symptoms.

The neurasthenic can always be reasoned with and be made to realize that his fears are groundless, although he may not always be able to dispel them.

Disturbance of sleep is a very common occurrence in neurasthenia. Many patients complain of inability to sleep, contending that they sleep for only a few minutes during the entire twenty-four hours; others have great difficulty in going to sleep and awaken early; some fall asleep soon after retiring, but awaken after a few hours and are unable to go to sleep again; in some the sleep is very light; marked restlessness occurs, and they

awaken upon the slightest noise; not infrequently they awake in a state of terror. Again the sleep is light and restless, and is disturbed by terrifying dreams; in practically all cases the sleep is unrefreshing, and the individual awakens feeling more tired than he was upon retiring.

Sensory Symptoms.—There is often complaint of a general tired feeling—that various portions of the body feel heavy; or of an unnatural or light feeling of some portion or of the entire body; or numbness, tingling, or burning may be complained of; that sensation is not acutely perceived, or that there is a slowness in its perception. Loss of sensation is not present.

Pain is also very frequently complained of. It may be diffuse or limited to some part or organ of the body, and is usually accentuated upon physical exertion or upon excitement. Headache and peculiar sensations about the head are common symptoms. The headache may be slight or severe and may be constant or occur only upon physical or mental exertion or as the result of emotional disturbances. It may be diffuse, or may involve principally the frontal region, the vertex of the head, or the occipital region; in the latter case the patient generally declares he has a pain at the base of the brain. The pain may be described as passing through the head from the back of the eyes into the occipital region; sometimes it is described as a pressure within the head, as if the skull were too small for the brain. Frequently there is a sense of constriction about the head, and at other times there is a feeling as if a weight were pressing on the top of the head. Backache is also frequently complained of, and there is a sense of weakness, dragging, pressure, and tenderness along the course of the spine. The tenderness along the spine is usually of a rather superficial character, and is relieved by deep pressure. The spine is generally more tender over its prominent parts and in the region of the coccyx; in this latter situation tenderness and pain are sometimes so intense as to interfere with sitting. Complaint is also made of many other disturbances of sensation, such as stiffness, soreness, a sense of heat or cold, etc., which may be referred to all or to any part of the body.

Many neurasthenics are hypersensitive to heat or cold, ex-

posure to slight increases in temperature giving rise to great discomfort.

Symptoms Pointing to Involvement of the Special Senses.—The visual centers may be either in a state of irritation, evidenced by the patient seeing spots, bright points, patches, etc., more or less constantly before the eyes, or there may be rapid fatigue of these centers, the individual complaining of inability to read for any length of time, the letters becoming after a short period indistinct, running into one another, and sometimes everything seems to be clouded and indistinct. If the eyes are used for any length of time there occur marked cephalic discomfort and a drawing sensation in the back of the head. The hearing may also become involved; thus the individual becomes sensitive to noises, complains of peculiar sounds in the ears, such as ringing blowing, whistling, etc.; in some cases both hearing and seeing are accompanied by discomfort, an apparent hypersensitiveness being present. In a few cases the sense of taste and of smell also become affected, the patient becoming either hypersensitive or complaining of a decreased acuteness of these sensations. The pupils are usually very mobile, and are frequently widely dilated.

Careful examination fails to reveal any organic basis for these complaints.

Vertigo.—There is a sense of uncertainty and giddiness, and the patient experiences a feeling as if he were going to fall. These attacks are usually accompanied by a marked state of fear, and in some instances may so far influence the individual as to prevent him from going out alone.

Symptoms Due to Disturbed Activity of the Motor Functions.—Frequently there is complaint of weakness and of rapid fatigue, but practically never of paralysis. There is a lack of strength, and movements are carried out slowly. Labor requiring physical effort is accomplished only with difficulty. Owing to the sense of weakness and rapid fatigue, patients fear that the weakness will develop into a paralysis.

Tremor.—Along with the sense of weakness and rapid fatigue there is often tremor of a rapid, fine, vibratory nature. Frequently this occurs only during voluntary movements and after

excitement, disappearing entirely while the individual is at rest. A fine fibrillary tremor affecting the orbicularis palpebrarum and the orbicularis oris muscles frequently occurs during emotional disturbances.

The tendon reflexes and the mechanical excitability of muscles and nerves are usually exaggerated. The muscle tone and volume remain normal, except in those cases in which there is much general emaciation, when the muscles may become flabby and the tendon reflexes be elicited only with difficulty. Loss of a tendon reflex never occurs in neurasthenia. Such loss always indicates the existence of an organic disease.

Cardiac Symptoms.—Attacks of cardiac palpitation accompanied by precordial distress are of frequent occurrence. Such attacks may come on after physical exertion or excitement, or may occur without apparent cause. Attacks simulating angina pectoris, in which there are diffuse pain in the chest and arms, rapid pulse, and marked anxiety, also occur. Complaint is frequently made of the consciousness of the heart's action, the individual feeling as if the heart were about to cease beating.

In neurasthenia the pulse-rate usually ranges from 90 to 120, acceleration occurring upon physical effort or as the effect of emotional disturbances. Frequently the taking of food or the use of coffee or tobacco, as well as sexual excitement, emotional disturbances, etc., will markedly increase the pulse-rate. In some cases pressure over a painful point accelerates the pulse-rate 10 or 20 beats a minute; this is known as Rumpf's sign. The blood-pressure in neurasthenia is usually low.

Vasomotor disturbances are present in a large number of cases, and consist of a feeling as of a rush of blood to the head, which may be associated with pulsation and peculiar sensations in or under the scalp; flushing of the skin, which may be localized or diffuse, and which generally is objectively evident, etc. The temperature of the skin in the flushed areas is usually slightly elevated, and slight irritation of the skin may cause severe and persistent redness, resulting in dermographia, urticaria, etc.

There may be rapid alterations of vascular tone—sometimes there is a more or less persistent venous congestion, the face

being flushed, and the lips taking on a bluish hue. The extremities are cold, moist, somewhat swollen, and cyanotic in appearance.

In women disturbances of menstruation are common; thus the period may become too frequent, the flow be excessive, or the interval prolonged and the flow scanty. An exaggeration of many of the nervous symptoms usually occurs at the menstrual period.

A very common symptom is that of consciousness of the throbbing of the aorta, and pressure over this vessel usually causes considerable pain.

Disturbance of Secretion.—There may be marked increase of perspiration, the entire body being moist; in some cases the hands and feet are constantly moist. The flow of saliva and of urine may be greatly increased. The secretions of the stomach, intestines, joints, etc., may be increased or decreased, and their composition may be altered. These changes may vary from time to time.

Symptoms Referable to Disturbed Activity of the Genito-urinary Apparatus.—In many instances there may be physical derangement of the sexual apparatus. This may be of prolonged duration, and when symptoms pointing to the genital organs are present, an organic condition should be carefully sought for. Early or excessive masturbation may be the causative factor of a neurasthenic state, or it may in itself be a symptom of a neuropathic disposition. At first masturbation is indulged in for the physical sensation induced, the sensation resulting from the local physical stimulation being sufficient to produce the desired result. Later mental imagery is called upon to assist the local physical sensation. The mind, being more or less constantly occupied with sexual thoughts, or as the result of excessive sexual indulgences, the genital centers remain in a state of abnormal activity, and there results an excessive secretion of the sexual glands. As a consequence erections occur readily, and emissions are frequent; finally erections become imperfect, premature ejaculations occur, and the patient observes that there is a loss of semen after urination or defecation. In these cases ejaculation is not accompanied by erection or

pleasurable sensation. As the result of these symptoms the patient becomes alarmed, depressed, and fearful of the occurrence of spinal cord diseases and of impotence.

In some cases, even without masturbation or excessive sexual indulgence, there is a gradual decrease of the sexual appetite and of sexual ability. Under these conditions the patient becomes depressed and alarmed, fearing total sexual incapacity. This fear of becoming impotent may interfere with erection and psychic impotence may result, which in turn may be followed by many neurasthenic symptoms. Sensory disorders, such as numbness, coldness, pain, sensitiveness, etc., may affect the genital organs. A mental state of fear, combined with depression, is most marked in these individuals.

The functions of the bladder are often markedly affected in neurasthenia. Owing to the concentrated and disturbed composition of the urine there may be irritation of the bladder, with frequent micturition. In other cases in which there is no local disturbance of the bladder frequent micturition may also be present.

The urine may be decreased in amount and highly colored, or there may be a marked increase in the quantity secreted; in either case there may be an increased frequency of urination. As a rule, the urine contains an increased amount of phosphates and oxalates.

Symptoms pointing to disturbance of the functions of the gastro-intestinal tract occur very frequently in this disease. Loss of appetite and peculiar sensations are complained of. The latter consist of a feeling of emptiness or of fulness; a sensation of constant hunger, which is not relieved by food; a quivering, trembling, or throbbing sensation referred to the stomach; pain on pressure over the abdomen; eructation; vomiting; meteorism, etc. All or any combination of these symptoms may be present. In many instances the individual complains of digestive disturbances, asserting that he is unable to digest acid fruits, meats, milk, etc. Many patients adhere to a very rigid diet, believing their condition to be due primarily to a gastric disease. Some individuals have so restricted their diet that they practically live on crackers and water. In some cases the patient

complains of distress immediately after eating; in others the gastric symptoms do not occur for an hour or more after taking food. The gastric symptoms are frequently accompanied by a sense of heaviness and of oppression, with palpitation, general weakness, and in some cases considerable abdominal pain. These symptoms usually become intensified after physical exertion, anxiety, or emotional disturbances. An examination of the gastric secretion shows no constant deviations—in some instances there is a hyperacidity; in others there is a hypoacidity or an anacidity, and in many there are variations of the secretion from time to time.

Constipation, peculiar feelings, and distress in the intestines are also frequently complained of. Attacks of diarrhea causing great weakness may occur; such attacks sometimes occur only in the morning, whereas others follow excitement and emotional disturbances. In one form of persistent diarrhea there is considerable pain, and a large amount of mucus in stringy masses is passed; in these attacks there is great exhaustion, and the patient regards these frequent stools containing this stringy mucus with great apprehension. The pain is very severe, and the attacks are dreaded. This condition is described as *mucous colitis*, and may persist for many years. The attacks are always increased by physical exertion, excitement, and emotional disturbances.

The gastro-intestinal disturbances cause considerable discomfort, and arouse in the patient's mind a fear of a serious disease of the gastro-intestinal tract. As these symptoms are associated with the taking of food, or are the result of the loss of appetite, the individual gradually restricts his diet or diminishes the amount of food taken, and as a consequence it is not unusual for loss of weight and strength and unhealthy conditions of the skin to occur. In many persons gastroptosis and enteroptosis occur as a congenital condition, or are the result of absorption of the mesenteric fat.

Trophic Disorders.—Trophic disorders are not common, but premature graying of the hair frequently occurs. The general state of nutrition may or may not be disturbed. In many cases in which there are much loss of sleep, restlessness, gastro-

intestinal symptoms, and the like there is marked loss of weight and strength.

Course of the Disease.—In the vast majority of cases neurasthenia develops very slowly, new symptoms appearing from time to time. The disease may manifest itself in early childhood, such children always presenting a marked congenital predisposition. This predisposition is manifested either by the history of neuropathic taint in the ancestry or by physical impairment of the health of the parents preceding conception, or by nervous debility of the mother during the time she was carrying the child. These children are usually nervous, and when overstimulated, either mentally or physically, complain of many neurasthenic symptoms. In older children or adults who are working under unfavorable mental or physical conditions a neurasthenic state may develop as the result of trauma, emotional shock, or acute illness. More often symptoms develop after physical or mental overexertion, particularly if combined with worry or if conjoined with unfavorable physical surroundings. The symptoms may be so mild at first that the individual does not heed the warning, or attributes them to a local physical condition, and hence does not apply the proper remedy. The symptoms gradually become more marked, the mental anxiety increases, the capacity for work diminishes, and, as a consequence, a vicious circle is established in which worry, introspection, anxiety, and decreased capacity for work are present, with increased demands, owing to the fact that the work is never completed.

If untreated and if the causative factors are not removed neurasthenia displays a tendency to grow gradually worse, and may last many years. Rarely are all the symptoms of this disease present, but usually some group of symptoms stands out prominently, which has led some authorities to describe the disease under the heads of the different types, for example: Gastro-intestinal, spinal, vasomotor, sexual types, etc.

Neurasthenia does not run a regular course. The course of the disease is marked by periods of improvement and exacerbations of symptoms; for example, there may be periods during which the patient feels comparatively well, and these

may alternate with periods of marked increase of symptoms. As a rule, the individual feels worse early in the day.

Prognosis.—Neurasthenia developing in early childhood, if promptly and properly treated, recovery is to be expected. As, however, these children are usually reared in an unfavorable atmosphere and are inherently weak, relapses are prone to occur. In some cases, particularly in those developing very early in childhood, a more or less chronic state exists, and the patient remains an invalid during his entire life. In these latter cases the symptoms change, however, from time to time; thus during one period gastro-intestinal symptoms may be prominent, whereas at another period cerebral symptoms, spinal symptoms, or sexual symptoms may be most marked.

The shorter the period during which the individual has been exposed to the harmful influences of overwork and worry, and the better the previous health, both mental and physical, the more favorable is the prognosis.

The vasomotor and sexual symptoms are the most difficult to eradicate. The prognosis, as a rule, for recovery from an attack is fairly good, provided the cause can be removed and proper treatment instituted. After one attack has occurred there is always danger of another if the patient is living amid unfavorable surroundings.

Attacks of neurasthenia occurring after an acute illness the result of resuming work before the mental and physical vigor has been reëstablished, if properly treated, usually present a good prognosis.

The cases of neurasthenia in which phobias are among the prominent symptoms are usually of prolonged duration and very resistant to treatment. Neurasthenia may frequently be combined with hysteria, and under these conditions the disease will run a prolonged course, and may be resistant to treatment. True insanity rarely if ever develops from a true neurasthenia.

Traumatic neurasthenia presents a somewhat unfavorable prognosis, and when combined with the anxiety attendant on law-suits, may tend to grow worse. Even in the absence of

this latter factor many symptoms may persist for years and prove very resistant to treatment.

Diagnosis.—In diagnosing this disease it is very important to remember that there are many organic diseases that are ushered in with neurasthenic symptoms, and also that the latter may occur during the course of an organic disease. It is, therefore, most important to make a careful physical examination, bearing in mind that mental fatigue, difficulty of thought, etc., may be among the early symptoms of a general paresis; that malignant growths, general arteriosclerosis, nephritis, diseases of the heart, chronic poisoning, etc., may all give rise to neurasthenic symptoms. The diagnosis of neurasthenia is, therefore, to be made only after a most careful physical examination, in which these diseases are excluded. The history of the case will often aid the diagnosis. On the other hand, it is just as important not to treat a neurasthenic individual for organic disease merely because symptoms are present that point to disturbed activity of some organ or organs of the body.

Neurasthenia is differentiated from psychasthenia by the fact that in the latter the mental symptoms are unduly prominent, and that although the patient realizes the absurdity of his feelings, yet he is unduly influenced by them, and reacts to them with marked emotional disturbance.

	Neurasthenia.	Involutional Melancholia.	Depressed State of Manic-depressive Insanity.
Age:	Childhood or early adult life.	After the fortieth year.	First attack usually between the fifteenth and thirtieth year.
History:	Of overwork and worry.	Indefinite.	Of previous attack of excitement or depression.
Facial expression:	Depressed or anxious.	Depressed and sad.	Depressed and sad.
Motor functions:	Rapid fatigue.	Usually restless.	Psychomotor retardation.
Judgment:	Good.	Delusions of persecution and of a somatic nature.	Delusions in which self-accusation or seeming unworthiness plays a part.

In differentiating neurasthenia from hypochondriasis it is necessary to remember that in the latter condition the in-

dividual's idea as to the state of his organs and as to the cause of his symptoms are fixed, and usually unreasonable and absolutely uninfluenced by argument. Symptoms pointing to mental or physical exhaustion play but a minor part in the picture of hypochondriasis, whereas disturbance of somatic sensations are of the utmost importance in diagnosing this disease. For example, when the patient experiences shortness of breath or has a cough, he will insist that his lungs are entirely destroyed; or in the presence of very minor symptoms he will assert that his kidneys or stomach are the seat of incurable disease, and he will persist in his belief in spite of reassurance and thorough examination by competent physicians.

Neurasthenia must also be differentiated from hysteria because of the marked difference of treatment indicated in these two diseases. In some cases a neurasthenic state develops in a hysteric individual. For the differential diagnosis between these two diseases see Hysteria.

The importance of careful history taking cannot be too strongly impressed upon the examiner, and in making the physical examination every effort should be made to discover the presence of mental disease or of organic physical disturbances. This is most important, so that the patient be not treated for neurasthenia when an organic physical disorder or a mental disease exists; and, conversely, that an individual be not treated for a physical ailment or a mental affection when a true neurasthenic state is present.

Treatment.—To prevent the development of neurasthenic states in children or in individuals who are of a nervous temperament is as important as the cure of the condition once it has set in. Children who are of a nervous temperament should not be allowed to study too hard at school; their life should be so ordered that they will spend a large part of the time out-of-doors playing and exercising. There must be supervision of their physical efforts in order to avoid undue fatigue. They should sleep in well-ventilated rooms, be given non-stimulating nutritious food, and regularity in habits as regards playing, working, eating, resting, etc., should be cultivated.

The bowels should be regulated. A careful examination should be made and all physical abnormalities should be corrected. Thus adenoids, enlarged tonsils, or an adherent foreskin should be corrected by surgical means. The child should be encouraged to exercise control over his emotions, and to cultivate a calm temperament. Care should be taken to prevent the formation of vicious sexual habits. The companions and friends of the child should be carefully chosen, and the studies should be supervised by a teacher who is temperamentally suited to the work. As the child grows older, or in the case of young adults, physical ailments should receive prompt attention. The nutrition should be supervised, and good mental and physical habits be acquired. Somewhat more than the normal amount of physical rest should be taken, and mental or physical overwork and excitement should be avoided. An occupation should be chosen that is agreeable to the individual, and that will not require efforts beyond the normal capacity of the patient. The same caution as regards the avoidance of overexertion, either mental or physical; the cultivation of good habits; the avoidance of excitement; the taking of the required amount of rest; prompt attention to all physical ailments, and the correction of minor physical disabilities should be insisted upon in all individuals who show a tendency to abnormal fatigability.

In the treatment of this condition after it has occurred the first and most important factor is to make a careful and thorough examination to discover if any organic disease is present. If the diagnosis of neurasthenia has been made, the most effective agent at our disposal that is capable of restoring the individual to his normal state is rest; rest must be physical and mental, in order to bring about beneficial results. The amount of rest, and what constitutes rest for different individuals, vary in different cases.

In markedly debilitated men, in many children, and in almost all women rest in bed is to be advised. In men who are accustomed to leading an active life, and who are not severely debilitated physically, rest in bed is inadvisable, as these patients are in a state of constant mental rebellion.

The food should be light and nutritious. Hydrotherapy,

electricity, and massage have been found to be beneficial. The bowels must be kept regular, and water taken freely. The surroundings of the patient must be pleasant and the room well ventilated. In most cases it is important to have a competent, tactful nurse, and to exclude the family and friends. Tonics and laxatives when required are indicated.

In my experience the most effective form of treatment, and the one that yields the promptest and most satisfactory results, is the Weir-Mitchell rest cure, carried out in a properly equipped hospital or sanatorium. The following is an outline of such treatment, and should be modified to suit the individual case:

A glass of cold water. Calisthenic exercise, five minutes; deep breathing exercise; Swedish movements, five minutes.

Scotch douche—patient to stand in warm water; sponge entire body alternately with hot and cold water; rub down with coarse towel.

Breakfast: May have stewed fruit—stewed or baked apple, cranberry sauce, pineapple; cereal, any sort, with plenty of cream; eggs prepared in any way; toast or bread—plenty of butter; milk or cocoa. Eat slowly, masticate well.

Rest one hour after breakfast.

Exercise, diversion, or occupation.

Glass of cold water; nourishment.

Rest.

Dinner: Must not have the same dish two days in succession. May have broth, meat, chicken, or squab; vegetables—potatoes, carrots, spinach, peas, beets, cauliflower, celery, string-beans; potatoes may be boiled, riced, mashed, in the form of potato cakes, baked, packed in half-shell. Salad—fruit salad, plain lettuce (no dressing), asparagus, beets; bread and butter, half-pint of milk flavored with tea.

Rest two hours after dinner.

Glass of cold water; nourishment.

Diversion, occupation, exercise, or general massage.

Rest.

Supper: Chops or other meats or eggs; sweetbreads; vegetables—potatoes, mashed, baked, or any style which patient did not have at dinner; one other vegetable which patient did not have at dinner; toast or bread and butter; light desert or stewed fruit; milk or cocoa.

Rest.

Exercise, diversion.

Cold pack, hot pack, or salt rub—use table salt, which is to be heated; moisten hand, take a handful of salt, and rub entire body, beginning with feet, and rubbing always in the direction of the venous circulation; abdomen is to be rubbed with a circular motion, beginning in lower right quadrant.

Cup of hot milk before retiring.

Sleep in well-ventilated room; bowels to be kept well open; avoid coffee, tea, tobacco, alcohol, fried foods, and heavy pastries.

If patient is poorly nourished, the amount of milk is to be increased until two quarts or more are taken daily. It should be given preferably immediately after meals. Sodium citrate may be added to the milk—5 grains to each $\frac{1}{2}$ pint of milk.

PSYCHASTHENIA

In this condition there exists an abnormal state of the mind in which the individual is governed by ideas which obtrude themselves more or less persistently upon the consciousness, and which may or may not be constantly present. These ideas have marked effect upon the emotional tone of the patient, usually giving rise to a more or less persistent state of anxiety, with some depression. As a general rule the patient realizes the falsity of his conceptions and the abnormality of these ideas, struggles against them, but is unable to prevent their occurrrence, or to subvert the overwhelming influence they exert upon his emotions, feelings, and actions.

The true psychasthenic is not insane, for he realizes the

absurdity of his ideas. Illusions, hallucinations, or delusions are not present in psychasthenia.

Etiology.—Heredity plays a most important part in this disease. In the great majority of cases there is a history of nervous or mental troubles, or of a chronic debilitating disease in the immediate ancestry. Many of these patients also show stigmata of degeneration. Women are affected somewhat more often than men. The disease develops most frequently between the twentieth and the fortieth year.

The same predisposing and exciting causes that are present in neurasthenia are active also in this condition. Among the exciting causes are excessive fatigue, sexual or alcoholic excesses, trauma; the periods of puberty, adolescence, and the climacterium; sensations connected with the heart, stomach, genital organs, vertigo, etc., which are accompanied by fear of serious disease; strong religious emotions, fright or fear, grief, prolonged worry, great anxiety, etc. Marked emotional disturbances, either sudden or prolonged, which are characterized by a disagreeable tone and a sense of fear, in a predisposed individual, serve as a most prominent exciting cause.

A thorough examination of patients suffering from psychasthenia, and a careful investigation as to the cause of some of the symptoms, will undoubtedly convince one that Freud's theory of psychic trauma, whether or not of a sexual nature, with consequent repression and submergence into unconsciousness, explains the underlying cause of this condition in some cases.

Symptoms.—The emotional state of the psychasthenic is always one of more or less persistent anxiety. These individuals are apprehensive, and are worried as to the outcome of their condition. Sensations referred to the various organs are accompanied by marked mental distress. Thus those who have palpitation of the heart or a sense of pressure in the epigastric region are fearful of sudden death or of attacks of angina pectoris; those who have pain in the head, that some brain disease exists; those who have attacks of vertigo, that they are going to suffer a stroke of apoplexy, etc.

Persistent morbid fears and emotional obsessions are among

the most common symptoms. These patients complain of a sense of discomfort, fear, great apprehension, and anxiety under certain conditions. They realize the groundlessness of their fears, and yet are incapable of self-control or of governing their actions. The individual may be afraid to go out alone (monophobia), never leaving the house unless accompanied by some one; he may fear to go into confined places such as theaters or churches (claustrophobia); may dread to touch a knife for fear of cutting himself; may be afraid to go up and down stairs for fear of falling; there may be a fear of dirt (mysophobia); a fear of open places (agoraphobia); a fear of darkness (nyctophobia), etc.

The affected individual realizes the unreasonable nature of these fears, and yet is powerless to prevent their disagreeable effect or to exercise control of himself when this fear exists. These states of fear are frequently accompanied by physical symptoms; thus the individual becomes pale, trembles, and breaks out into profuse perspiration; the pulse becomes rapid, and sometimes diarrhea or vomiting attacks occur. In the more severe cases, unless relief is afforded, all the evidences of collapse are present.

Imperative Ideas.—Ideas that are not produced by association, but start up independently and intrude themselves upon the thoughts so forcibly that they cannot be banished, although they are recognized by the patient as being foreign and strange to his mental personality, are termed imperative ideas.

Ideas foreign to one's line of thought often intrude themselves upon the consciousness of normal individuals, but in this case they are quickly dispelled, and do not cause emotional disturbance; in the psychasthenic, on the other hand, these ideas intrude themselves upon the consciousness, cannot be banished, and cause emotional disturbance. Thus, for example, while at prayer, thoughts of sacrilegious or of an obscene nature may intrude themselves upon the consciousness of the patient, and cannot be banished, occasioning a disagreeable emotional tone; or, while he is planning some pleasure or thinking of those in whom he is interested, the idea of death or of accident enters persistently and prominently into his consciousness.

Intellectual Obsessions.—In this condition there occurs a mental state in which the patients are more or less constantly in a state of doubt—it is impossible for them to reach a conclusion, even in the most trivial matters, their entire time being occupied with self-questioning and indecision.

In severe cases the individual is so dominated by his obsessions that he is practically unable to devote any time to the duties of life. The mental state of these patients is distressing: they realize the uselessness and absurdity of their indecision, and yet cannot resist the mental processes and actions which it occasions. These patients are more or less constantly in a state of fear and anxiety, and the most trivial acts of life are carried on only after a prolonged effort and indecision. To this group of symptoms belongs the *folie du doute* of the French: the patient, upon arising, spends hours in dressing, and it is only with the greatest difficulty that he is able to make up his mind which tie to wear or which suit to put on, dressing and undressing, until finally half of the day may be passed in indecision; or if he must copy a document, he spends hours in going over the same for fear of an omission; after retiring for the night he will get up repeatedly to be sure that he has locked the doors or turned out the lights.

The uncertainty and indecision are accompanied by fear, anxiety, and sometimes by such physical symptoms as cardiac palpitation, profuse perspiration, trembling, weakness, dilatation of the pupils, etc.

Morbid Impulses—Volitional Obsessions.—In this condition the individual is deficient in will, being unable to restrain himself from performing certain acts. He feels an uncontrollable desire to perform some act, which may be unconventional, dangerous to himself or others, or useless. While obsessed by this impulse he suffers from mental discomfort and anxiety, which are followed by a sense of relief after the performance of the act. As examples of such acts may be mentioned a feeling that he must count every animal he sees; must skip certain words in sentences; must touch certain objects. At other times these impulses assume a more serious character, and the patients feel that they must take objects they admire;

under this group come true kleptomania and dipsomania. Others feel impelled to throw objects at people, etc.

Among other symptoms that occur less frequently are states of consciousness in which the patient feels as if he were not himself—he is obsessed by a feeling of strangeness, as if his attitude toward his surroundings had been altered. In more marked cases there is complete change of personality, and the individual no longer recognizes himself or his original surroundings; the past becomes completely obliterated, and he possesses himself of an entirely new personality, and reacts differently from his original self to his surroundings. This state may last for from a few hours, days, weeks, or months to many years, and when he again recovers his original personality he has no recollection of the actions, persons, and events that occupied him during the period when he was in a state of abnormal consciousness.

The psychasthenic may develop along with the neurasthenic state, or it may occur in an individual suffering from neurasthenia; it generally develops independently of this disease. Psychasthenia may also be complicated by or complicate hysteria.

Course.—This disease usually begins gradually, but may be of sudden onset, following some emotional disturbance or injury. A patient who develops this disease shows many peculiarities early in life: he is, as a rule, peculiar, scrupulous, and has difficulty in reaching conclusions; such patients are sensitive, many are eccentric, and all are very emotional and impressionable.

In cases in which the symptoms are of slow onset the individual himself will notice that there is a certain strangeness about his mental activity; that his physical sensations and his mental processes are accompanied by a queer emotional change, and finally that physical sensations induce great anxiety and emotional disturbances which he is powerless to prevent. He realizes that his actions are governed by thoughts and impulses that come unsolicited and that he is unable to banish.

The disease is subject to remissions and exacerbations, the patient being at times much improved and almost free from

symptoms, whereas at others his life is made extremely unhappy by the persistent presence of many disagreeable symptoms.

Prognosis.—In many cases the outlook is serious, the disease lasting for years, and manifesting a tendency to the development of fixed delusions. On the other hand, some cases occur that are very amenable to treatment and that make a good recovery within a reasonable time. In still other cases, as the result of the persistent and constant presence of morbid impulses, fixed ideas, etc., the individual isolates himself and leads a life of seclusion and unhappiness. The more marked the psychopathic hereditary tendencies are, and the more unsuited the surroundings for carrying on the proper treatment, the more unfavorable, as a rule, is the prognosis.

Diagnosis.—The differentiation is made from *neurasthenia* by the fact that in neurasthenia the symptoms are those of fatigue, either mental or physical, whereas in psychasthenia fatigue symptoms are generally absent, and characteristic mental symptoms, whose true import the patient recognizes, are present.

From *hysteria* the disease is differentiated by the fact that in hysteria symptoms are present pointing to abnormal physical functions, such as paralysis, contractures, disturbances of sensation, attacks of varying character, intervals during which the individual feels perfectly normal, whereas in psychasthenia there is an absence of these symptoms with a prominence of the characteristic mental symptoms.

In some individuals there may be an association of neurasthenic and psychasthenic states, or of hysteric and psychasthenic states, or all three conditions may be combined.

From *true insanity* psychasthenia is differentiated by the fact that in the latter the individual clearly perceives his abnormality, and often realizes the absurdity of his symptoms, whereas in insanity the patient is incapable of realizing the true import or absurdity of his symptoms.

Treatment.—A careful examination must be made so as to exclude all organic diseases and in order to gain the individual's confidence. It is well to explain frankly to the patient the

nature of his disease, and to secure his coöperation in the treatment. All physical defects should be corrected, and the general physical condition of the patient looked after, in the same manner in which the neurasthenic is treated.

In some individuals suitable occupation, diversion, and suggestion are sufficient to bring about marked improvement or cure. In others, in addition to occupation, diversion, rest, etc., a system of gradual mental and temperamental reëducation should be begun. In still another group of cases good results can be obtained only by means of psychanalysis, laying bare the submerged irritants, and bringing them again into the consciousness, together with the eradication of the mental traumas that are at the root of the psychasthenic state. In many cases one must use all these means to bring about a favorable result.

The treatment must usually extend over a prolonged period, sometimes lasting years. It is most important, in the treatment of this condition, that the patient have some suitable occupation or diversion which should occupy much of his time; physical work, such as farming and mechanical labor, painting, drawing, etc., are all to be advised.

Treatment by psychanalysis is applicable only to a limited number of cases; these should be selected only after a very careful consideration as regards the possible harm that may be done, and should be carried out only by those who are experienced in this line of therapy.

ANXIETY NEUROSIS

In almost every form of psychoneurosis a state of mental anxiety is more or less constantly present to a greater or less degree, but this anxiety is usually secondary to fear which is aroused by the presence of other symptoms.

A condition is occasionally seen in which the general state of anxiety is constantly present, and which is associated with all the mental and physical activities of the individual. Such persons are not true psychasthenics, hysterics, or neurasthenics: they live in constant anxiety, placing a pessimistic interpretation

on all abnormal sensations or occurrences. They are usually depressed, and show a general irritability. As in the other psychoneuroses, the chief cause of this condition is hereditary taint. The course of this neurosis is chronic. The outlook for recovery is very gloomy, as it is difficult to alter the temperament of these individuals. Prolonged treatment may bring about some improvement. In some cases psychanalysis relieves the causative psychic trauma, and may be followed by improvement or recovery.

OCCUPATION NEUROSIS—OCCUPATION SPASMS

This condition is characterized by the occurrence of pain, weakness, tremor, or cramp, or any combination of these states, in certain muscles or groups of muscles. The symptoms appear only when these muscles are called upon to perform some accustomed occupational movements, but do not occur when these muscles or groups of muscles perform other movements than those connected with the accustomed occupation.

The more delicate and highly differentiated the movements that enter into the performance of the occupation, the more readily does the overuse of these muscles set up this condition.

It occurs more frequently in the upper extremities, but may affect any part of the body. The most common forms are those connected with writing and the occupation of telegrapher, typist, pianist, violinist, trumpet-player, dancer, seamstress, cutter, blacksmith, milker, etc.

Etiology.—In the vast majority of cases a neuropathic heredity is present. Many of these individuals are also neurasthenics. Not infrequently the condition occurs in more than one member of a family. While the neurosis itself is not directly transmitted, a tendency toward either local or general fatigability of the nervous system may be inherited, which evidences itself when there is general or local overuse of the muscles under unfavorable conditions. Any occupation that requires repeated, continuous, and prolonged movements which are limited in extent and which are of a purely voluntary character, when carried on under unfavorable conditions in a predisposed individual, may be followed

by the development of this condition. It may also occur after injury or following any debilitating disease. It occurs more frequently in young adults, but may be present in childhood. It is also more likely to occur when work is carried on under physical or mental strain.

Pathology.—Careful examination has failed to reveal organic changes in either the nervous or the muscular system, and this condition must, therefore, be classified with the functional neuroses.

Symptoms.—The disease is usually insidious in onset. At first the individual notices that the movements connected with his occupation are carried on with some little difficulty or discomfort, which occurs only at intervals, and usually only when his attention is directed toward the muscular movements incident to his occupation, or when extra effort is demanded of him. The distribution of the symptoms is dependent upon the occupation: thus in writers' cramp, pianists' cramp, seamstresses' cramp, milkers' cramp, etc., the hands and fingers are involved; in blacksmiths' cramp the shoulder muscles are affected, and in dancers' cramp the calf muscles are involved.

The condition may manifest itself in several ways: in the most common form the individual notices that he tires more readily than usual, and that he cannot carry on his occupation with his occustomed speed; also that there is hesitation in his movements, which interferes with his work. Gradually, in the course of his occupation, spasms of the muscles develop.

At first, in spite of this spasmodic condition, the patient can continue his occupation, but gradually the difficulty increases, and any movement connected with his occupation occasions a spasm. The more he thinks about his work, and the greater the effort he puts forth to continue his work, the more marked does the interference become.

If this disease attacks writers, the spasm affects the flexors of the fingers, especially those of the thumb and the index-finger, and gradually extends to the muscles of the hand and forearm. In attempting to write, the letters are irregularly formed, interrupted, and very uneven in size. In other occupations the same disturbances take place: at first the spasms are

mild in character and limited in distribution; if, however, the occupation is continued and the attention is centered upon the effort to carry on the work, the spasms increase in severity and the distribution becomes more extensive.

Spasm is not the only manifestation of the neurosis: in some individuals, instead of spasm, tremor develops, a combination of spasm and tremor being somewhat common. In another group of cases instead of spasms a simple weakness becomes evident; this is known as the paralytic form, and is quite uncommon. If the occupation is persisted in the necessary movements finally become impossible, and when an effort is made to perform these movements, a relaxation of the muscles occurs together with an inability to cause a voluntary contraction to effect the necessary movements.

A sensory or neuralgic form of occupation neurosis is also encountered. In the spasmodic form pain very frequently attends the muscle spasm, giving rise to a true cramp; but in the neuralgic form the occupational movements may cause pain, which in itself may be so severe as to interfere with the carrying on of the occupation, there being a complete absence of muscle spasm.

The objective examination, as a rule, yields a negative result. The tendon reflexes are often increased, but there is no disturbance of sensation or motion and no tenderness; the symptoms manifest themselves only upon the effort to perform the movements that are concerned with the individual's occupation.

Course.—The course of the disease is usually slow; the onset is gradual, and the condition persists for a long time. The prognosis is not very favorable, for even after recovery has taken place relapses frequently occur. In the sensory form the prognosis is better. The longer the duration of the disease, the more unfavorable is the prognosis.

The diagnosis is usually made without difficulty. Care should be exercised that the onset of an organic disease is not overlooked. Occupation neurosis must be diagnosed from hysteric states in which the patient is unable to follow his usual vocation; as a rule, there is little difficulty in making the differentiation. The presence of other symptoms of hysteria and a careful consider-

ation of the history of the onset and the progress of the condition usually enable one to arrive at a correct diagnosis.

In the hysteric and neurasthenic states the difficulty or inability to carry out local voluntary movements is not limited to the special movements required in a certain occupation, but occurs with all voluntary muscular contractions.

Treatment.—The patient should be promptly advised to discontinue the occupation. The individual's general health should be looked after. Massage and special calisthenics are of great benefit; the patient should be instructed to carry out all movements in a free and graceful manner, avoiding all cramped positions. If the occupation is again resumed, the necessary movements should be carried on under different conditions and in a different manner than they were at the time the individual was affected.

Drugs are of little value. Special forms of apparatus are made that afford support to the muscles in certain forms of occupation neurosis; this is especially true in the case of writers' cramp, for which a special splint to support the wrist and fingers is used.

NEUROSIS CHARACTERIZED BY SPASMODIC MUSCLE CONTRACTIONS

Localized Muscle Spasms.—Under this head will be considered those abnormalities of movement that are limited to a muscle or to a group of muscles which are supplied by one or more nerves, the contraction being due either to irritation of the nerves themselves or reflexly to centers in the anterior horn or the higher cerebral centers. Such muscle contractions are not necessarily purposive in character, nor is it always possible to reproduce them voluntarily. They differ from the tics in that the latter are the result of contractions of groups of muscles whose combined action brings about a voluntary or purposive act. The tic can always be reproduced voluntarily.

FACIAL SPASM

Spasmodic contraction of the muscles supplied by the facial nerve or nerves is probably the form seen with the greatest frequency.

Etiology.—This condition occurs generally in adult life, and more frequently in women than in men. As a rule, a neuropathic disposition exists in the individual. The condition has been known to occur after sudden emotional shocks. It is a well-known fact that muscle contractions occur as the result either of a reflex sensory irritation or of a voluntary impulse, or it may be the result of some involuntary coördinative activity. It is in consequence of the uninhibited activity in the first-named condition that facial spasm occurs; consequently the commoner causes of this condition are sensory irritations in the region supplied by the fifth nerve, such as irritative conditions in the nose, throat, mouth, scalp, teeth, eyes, ears, etc.; refractive errors, local disease of the eyelids, etc., may also produce this condition.

Facial spasm occurs with special frequency in facial neuralgia. Irritative lesions in facial areas in the precentral convolution of the brain may cause spasmodic movements which start in the face and gradually extend until the entire side of the body is involved, thus giving rise to Jacksonian epilepsy. Tumor of the facial nerves or an irritative condition at the base of the brain may also give rise to facial spasms. Irritative affections of other parts of the nervous system may sometimes be the cause of facial spasm. Clonic twitchings of the muscles supplied by the facial nerves may sometimes precede a facial paraylsis.

Pathology.—In the great majority of cases facial spasm has no pathologic basis; in a few cases organic changes of an irritative nature may be present, occurring in the course of the nerve, in the middle ear, at the base of the brain, in the medulla, or may involve the brain itself.

Symptoms.—The principal symptom of this condition is a sudden, lightning-like contraction of the muscles of the face. The contraction occurs at intervals, or a series of contractions may occur rapidly, a longer interval existing between each paroxysm.

This disease is at first usually limited to one side of the face, but both sides may eventually be involved. Either all or some of the muscles supplied by the facial nerve may be affected. The muscle most frequently involved is the orbicularis palpebrarum, but the zygomatic muscles and the levator anguli oris are often affected along with the muscles of the eyelid. Sometimes the spasm involves the muscles of the mouth and chin, and in some cases all the muscles of one side of the face become affected. Occasionally, in addition to the clonic, lightning-like contractions, tonic contractions occur in which the muscles remain contracted for several seconds. Under these conditions the eyelids are closed, the forehead is thrown into furrows, and the nose and mouth are drawn to the side of the face affected. These muscle contractions are usually least marked during mental and physical rest, and are exaggerated and intensified by physical exertion, excitement, and any sensory stimulation of the fifth nerve or those nerves capable of reflexly stimulating the seventh nerve; thus the spasms increase when talking or

eating, on exposure to cold drafts, on efforts to examine the patient's eyes or throat, etc.

Vasomotor and trophic disturbances are rarely present. The spasm itself is never painful, and some individuals are not even conscious of these twitchings. The voluntary movements of the facial muscles are not, as a rule, affected, but the spasm may occur during these movements and disturb their course; in some cases efforts to move the facial muscles voluntarily bring on or increase the symptoms.

The tonic spasm that is limited to the muscles that close the eyes is termed blepharospasm; this may be unilateral or bilateral. The clonic form, known as blepharoclonus, is not uncommon.

Course.—This disease usually runs a chronic course. The spasms begin in a few muscles, and extend until the entire face is involved. The disease is especially subject to remissions; the intermissions or remissions may come on suddenly and last for variable periods of time, but there is a great tendency for the condition to recur.

Prognosis.—The prognosis is unfavorable. A few cases have been known in which, either spontaneously or as the result of treatment, recovery took place within a short time. If the condition has become firmly established, the prospects for recovery are very slight; even if intermissions should occur, the probabilities are that the spasms will recur and continue, with remission, throughout the patient's life.

Diagnosis.—This condition must be differentiated from an irritating tumor at the base of the brain, from Jacksonian epilepsy, and from facial tic. In the first two conditions the diagnosis is readily made from the occurrence of other symptoms, indicating the presence either of some irritative disease at the base of the brain or of an irritative condition involving the cerebral cortex. From *facial tic* the diagnosis is readily made when one remembers that a tic is a complex, purposive act which can be temporarily arrested, and which is often preceded by an impulse to perform this act, and which eventually cannot be resisted; that after the occurrence of the muscle contractions there is a sense of relief; that, on the other hand, facial spasm is a reflex contraction of muscles supplied by the seventh nerve,

the contraction coming on without warning. Generally, as the result of his efforts to avoid the contractions, they occur with greater frequency and increased severity; moreover, the movements of facial tic can be voluntarily reproduced by the patient, whereas the movements of a facial spasm cannot, as a rule, be voluntarily reproduced.

Treatment.—It is important, in these cases, to make a most careful examination, to discover, if possible, a focus from which a sensory reflex irritation could arise. In this examination one must keep in mind the fact that these sensory irritations, although present most often in the area supplied by the fifth nerve, may yet occur at some distance. The throat, nose, teeth, eyes, ears, scalp, etc., should be carefully examined, and if any evidence of abnormality is present, it should if possible be corrected. Careful examination should also be made of the respiratory, cardiac, abdominal, and pelvic organs, and abnormal conditions found should also be rectified if possible.

In cases where, after the correction of all physical defects, no relief follows, it may be advisable to attempt to ascertain if the irritation arises from some of the smaller filaments of the fifth nerve. For this purpose it is permissible to inject, at different times, the supra-orbital, the superior maxillary, and the inferior maxillary nerves with a weak solution of cocain. This is done for diagnostic purposes, to ascertain whether the spasm can be temporarily inhibited. If one is fortunate enough to discover the irritation in one of these branches of the fifth nerve, the subsequent injection of the affected nerve with alcohol, according to the method of Schlosser, may bring about permanent beneficial results.

In those cases that are not relieved by the foregoing procedure other measures must be used in the endeavor to give the patient relief. If the patient is of a neuropathic constitution, he should receive appropriate treatment in the form of rest, proper diet, exercise, hydrotherapy, etc. Hot applications to the face and the free use of sedatives are indicated. Bromids, cannabis indica, hyoscin, atropin, and, under some conditions, morphin may also be used.

SPASMS OF THE MUSCLES OF MASTICATION

Spasms limited to the muscles of mastication are not common. They may be of the tonic or the clonic type. Tonic spasms limited to the muscles of mastication may be a symptom of tetanus, meningitis, tetany, or epilepsy. They occur reflexly, as the result of an inflammatory condition of the temporomaxillary fossa or its neighborhood, irritative conditions in the mouth, such as an unerupted wisdom tooth, as an evidence of hysteria, etc. Clonic spasms of the masseter muscles occur in chills, hysteria, epilepsy, neuralgia, ear disease, etc.

The prognosis of the spasms of the masseter muscles is usually good if the cause can be discovered and removed. Treatment should be directed toward the discovery and removal of the cause.

SPASMODIC TORTICOLLIS

Spasms in the muscles of the neck may be unilateral or bilateral. They may be limited to individual muscles or to several muscles of one or of both sides. The muscles supplied by the spinal accessory nerve are especially likely to be affected. The spasms which may begin in the muscles supplied by one nerve do not, as a rule, show a tendency to become limited, but are apt to spread to other muscles during the course of the disease.

Etiology.—This disease is especially liable to affect individuals of a neuropathic or psychopathic constitution. A direct hereditary transmission is, however, rare.

Reflex irritations from the fifth nerve and occipital nerve, refractive errors, organic diseases of the brain, disease of the internal ear, chronic poisoning from alcoholic and metallic substances, infectious diseases, influenza, typhoid, pneumonia, mental or physical overexertion, shock, exposure, rheumatism, etc., are among the possible causes. The disease occurs more frequently in women than in men, and may appear at any time in life, although it occurs with greatest frequency after the thirtieth year.

Pathology.—No organic changes in the central or peripheral nervous system are found. According to views expressed by Brissaud and Oppenheim, this disease is due to a condition of

irritation in the nerve centers situated in the cortex of the brain, both in the motor and in the sensory zone that corresponds to the cervical muscles; these observers assert that the instability of these centers, which is mostly inherited or congenital, is the cause of the disease.

The symptoms depend upon the extent of the muscular involvement—whether one or both sides are involved, and whether the spasmodic contractions are clonic, tonic, or mixed. As a rule, the clonic twitchings are associated with a transient tonic contraction of the muscles. If the muscles of one side are affected during the contraction, the face may be turned toward the other side; the ear approaches the clavicle, and the chin is raised; as a rule, the contractions cause only a rotation of the head to the opposite side. If both sides are affected, the head is thrown backward, and there is a wrinkling of the forehead; again, the spasms may extend to the muscles of the shoulder and arm, in which case, when the head is rotated away from the side of the spasm, the shoulder is elevated and the arm is abducted. In some cases the spasmodic contraction may be so slight that the movements of the head are hardly noticeable; in other cases the contractions are so violent that the head is either thrown backward or rotated and thrown to the side with great force. In fact, these movements may be so constant and forcible that speaking, eating, and sleeping are all rendered difficult. The muscles affected may become hypertrophied. In some cases the spasmodic seizures are present almost continuously, occurring in paroxysms, with a tonic contraction as a termination. In these severe cases there are usually remissions during which the condition is not so severe. In some the spasmodic seizures occur in the form of attacks, separated by intervals during which the spasms are completely absent. The condition is aggravated by emotion, physical exertion, observation, etc. The spasms may cease entirely when the individual is in the recumbent position, and be present only when he is in the erect posture. In some cases the spasm can be temporarily arrested by making pressure upon certain points of the neck or head or by holding the chin, etc.

As a rule, pain is not present, but many of these patients

complain of an aching sensation in the neck and shoulders. There is no evidence of paralysis nor any disturbance of sensation. As a rule, the spasmodic contractions cease during sleep, but this is not invaraibly the case. Mental disturbances are sometimes associated with these spasms.

Occasionally depression and anxiety precede the disease, and in some rare cases the spasmodic seizures cease and mental symptoms appear, with the recurrence of the spasmodic condition after the mental symptoms disappear.

Course.—The disease is essentially chronic in its course. It is marked by remissions and intermissions that may last for variable periods of time. In some cases, owing to the difficulty in eating and to the interference with sleep, marked physical deterioration and loss of weight take place. The disease lasts for years. It attains a certain degree of severity, remains stationary, or may vary in intensity. The condition may become so distressing as to lead to attempts at suicide.

Prognosis.—The prognosis is extremely unfavorable. The younger the individual, the shorter the duration of the condition, and the less severe the spasms, the more promising is the outlook for recovery. In those cases in which some direct relation can be established between sensory irritation of the occipital or fifth nerve and the spasmodic torticollis, a more favorable prognosis is presented. In those cases that occur in individuals who present hysteric symptoms the prognosis is less favorable. The older the individual, the longer the duration, and the more severe the spasms, the graver is the outlook.

Diagnosis.—This condition is distinguished from *rheumatic torticollis* by the severe pain and tenderness of the muscles to pressure and by the pain on movement. Pain and tenderness are absent in spasmodic torticollis. From *congenital torticollis* it is differentiated by the fact that the condition is present from birth and that secondary changes occur in the cervical vertebræ, together with shortening of the muscle and permanent fixation of the head.

A persistent tonic torticollis should always suggest a local disease, *e. g.*, one involving the cervical vertebræ or the brain, such as a tumor or a diseased condition of the neck, etc.

Treatment.—A careful examination should be made to discover the cause of the affection, and if it is found, it should be treated. If the cause cannot be ascertained, the general health of the patient should be improved by diet, tonics, hydrotherapy, massage, etc. He should lead a life free from excitement, and follow an occupation that does not fatigue or unduly expose him to observation. Various forms of sedatives, such as the bromids, tincture of gelsemium, valerianate of zinc, asafetida, cannabis indica, etc., may be of benefit in relieving the spasmodic movements. Sometimes tincture of belladonna given in increasing doses to the physiologic limit may be followed by improvement. In more severe cases morphin, given hypodermically, is occasionally recommended. This drug must not, of course, be used unless it is absolutely necessary. Attempts to strengthen the opposing muscles by means of massage, systematic exercise, and electricity may be of benefit. Electricity, as a rule, has not given much satisfaction, but it may be tried. It is advisable to use the galvanic current, with the positive pole over the spinal accessory nerve and the negative pole at some indifferent point. Counterirritation by means of a plaster or the actual cautery along the back of the neck has in some cases proved beneficial. The application of mechanical supports, either in the form of a collar (a ring around the head fixed to a rod, which may be attached to the body or to the neck) or a pad that produces pressure upon a pressure-point, if one is present, may be followed by improvement.

Operative treatment of this condition has in some cases resulted in a temporary improvement. Careful consideration of the final result obtained in many cases leads me to the opinion that operative interference is not justifiable, since the final result is not good, and some cases have been rendered considerably worse.

Some authorities have found psychotherapy of benefit in this condition.

SPASMODIC AFFECTIONS OF THE MUSCLES OF THE TRUNK AND EXTREMITIES

These affections occur very infrequently, and then only as the result of organic disease of the brain or spinal cord or following their overuse in certain occupations. The spasms may affect either single muscles or groups of muscles, and may be tonic or clonic. They may involve the muscles of the upper extremities, of the lower extremities, of the diaphragm, of the trunk, of the pelvis, etc. When the spasms affect the muscles attached to the pelvis, they cause peculiar postures on standing and a very irregular gait. In extreme cases they bring about a tendency to walk on all four extremities, a condition that has been described under the title of "tortipelvis" and "dysbasia lordotica." In this condition the deformity is persistent, and consists of a marked lordosis of the dorsal lumbar region, the muscle twitchings being evident on walking or standing, but not while the patient is in the recumbent position. The spasmodic movements also often affect the upper extremities.

A careful examination in all these cases of spasmodic neuroses fails to reveal the presence of paralysis, sensory disturbance, etc., and we must, at least at the present time, acknowledge our inability to point out organic pathologic changes in any portion of the body, and also we must admit that mental treatment in some cases has brought about relief or cure.

TIC (HABIT SPASM)

Tics are movements that can be reproduced voluntarily. At first the movements are produced for some definite reason, and by their constant repetition a habit is formed, until they are finally reproduced unconsciously, involuntarily, and without apparent cause. These movements may increase in intensity and in frequency and be associated with other acts.

Tics can be controlled, at least temporarily, for a short time, but this usually causes a sense of distress and anxiety. As soon as the movements are allowed to go on the individual feels a distinct sense of relief. In some cases the movements are preceded by an unrest and by a feeling as if they must occur.

The spasms cease during sleep and when the mind is strongly diverted in another direction.

Etiology.—The disease occurs more frequently in children between the ages of six and thirteen. As a rule, there is a neuropathic tendency. The child may acquire these tics as the result of imitation or they may first be performed to relieve some physical discomfort. They have been known to develop from some slight irritation in the nose, eyes, mouth, or any other portion of the body, and to follow fright. Sometimes these habit movements develop without cause. Thus children whose hair is long may shake their heads backward and to the side in order to throw the hair out of the face; this movement may be continued even after the hair has been cut. Those who wear badly fitting clothes may, first as the result of discomfort, go through movements to adjust their clothing more comfortably; these movements may be continued even in the absence of the exciting cause, etc. In some cases these habit movements occur in epidemic forms, many individuals becoming affected. Although tic develops more frequently in childhood, yet it is often present in adults.

Pathology.—A most careful examination fails to reveal the presence of any organic pathologic change. In this disease the acts that are performed and that are abnormal under the circumstances correspond to normal activities under proper conditions; consequently one would not expect to find organic disturbances. The conditions under which these acts occur are the result of a deficient psychic inhibition which allows purposive activities to take place without either adequate reflex stimulation or adequate external, defensive, or volitional requirement.

Symptoms.—Any volitional act that is produced at first for some definite purpose, in imitation of the acts of others, or thoughtlessly, may, by constant repetition, become habitual and be reproduced unconsciously. The tic may consist of a single movement or of a number of movements, or may be general. The movements may occur occasionally, frequently, and sometimes almost constantly. In many cases the effort to overcome these movements is accompanied by marked mental distress, and great relief is felt when they are allowed to occur.

The tic may involve the eyelids, giving rise to winking of one or both lids; this may or may not be accompanied by wrinkling of the forehead, and may involve the face, causing grimaces to be made; these may be bilateral or unilateral. The head may be shaken from side to side as if to toss the hair from before the eyes; movements of the lips and tongue may take place, giving rise to sucking, sniffing, pouting, biting, gulping, etc. The shoulders and arms may be affected, causing shrugging movements, the arms being thrown from side to side, snapping of the fingers, etc. The trunk may be affected, giving rise to peculiar movements; or the lower extremities may be involved, giving rise to peculiarities of gait, etc. Tics of speech may occur in which the individual repeats words, or he may skip certain words in a sentence, etc.

Distraction and occupation, as a rule, have a soothing effect upon the tics, and excitement, emotional disturbance, observation, etc., tend to increase them.

Course.—The onset of this condition is usually slow. It frequently follows anxiety or some emotional disturbance. In childhood the onset may occur as the result of imitation, reflex irritation, discomfort, emotional disturbance, etc. The condition runs a chronic course, marked by remissions and intermissions. In a few cases there is a gradual extension of the tic from one part of the body to the other, and eventually mental failure occurs.

Prognosis.—Tic beginning in early life usually presents a good prognosis when the child can be placed under favorable conditions for treatment. In adults the prognosis is not so good, intermissions being frequently followed by recurrences.

Diagnosis.—The diagnosis is made from the fact that the movements that occur can be voluntarily reproduced; that these movements occur without the patient being conscious of them; that they are automatic; that the efforts to restrain them cause distress, and that relief occurs when they are allowed to recur.

This disease must be differentiated from *hysteria*, in which condition the movements come on suddenly and are associated with other evidences of hysteria. From *chorea* this disease is

distinguished by the fact that in chorea the movements are purposeless, occurring almost constantly, and are rendered worse when an effort is made to restrain them, whereas in tic the movements are purposive, frequently lengthy intervals occur between the movements, and occupation has a soothing effect; also they can be voluntarily repressed. It is well to remember that in some cases of *dementia præcox* peculiar purposive movements of the face and body occur.

Treatment.—In those cases which occur in children as the result of discomfort or of reflex irritation the exciting cause should, if possible, be removed. In all cases the physical condition of the patient should receive attention. In some cases it is necessary to isolate the patient from his family and friends. For the cure of the tic it is necessary to reëstablish self-control; this is done by reëducation, the individual endeavoring voluntarily to control and prevent the occurrence of the spasm. He should go through a regular exercise before a mirror several times a day, voluntarily contracting the muscles which are affected and also their opponents; this treatment is best carried on in the presence of some one who has control of the patient. In children rewards may be given for successful efforts to control the affection. Harsh treatment and punishment usually tend to aggravate the affection. Hydrotherapy and generally gymnastic exercises are also of benefit, in so far that they improve the individual's general condition and seem to exert a soothing effect upon the patient.

Anemic and debilitated individuals should be given tonics, and those of a nervous, restless temperament may be given sedatives.

CHOREA

Chorea is a term derived from the Greek word Χορεία, meaning to dance. Under this term are grouped a number of conditions that exhibit as their characteristic symptom involuntary, uncontrollable, unexpected, and purposeless contractions of muscles or of groups of muscles.

Under this head are included chorea minor, Sydenham's chorea or St. Vitus' dance; Huntington's chorea, or chronic

hereditary chorea; chronic progressive chorea of adults; chorea accompanying organic brain lesions; senile chorea; chorea insaniens; electric chorea, and Dubini's chorea. These conditions may be divided into several groups, which present a similarity of motor symptoms, but which should be correctly differentiated one from the other.

For purposes of description these conditions will be divided into four groups: Group 1 includes those forms of chorea that are probably due to an acute infectious or toxic condition, chorea minor, or Sydenham's chorea, and chorea insaniens; Group 2 includes senile chorea, chorea accompanying organic brain lesions, chronic progressive chorea of adults, and Huntington's or hereditary chorea; these choreas are due to a progressive organic degenerative cerebrospinal disease of unknown origin; Group 3 includes epidemic chorea, which is due to imitation, and is a form of hysteria; Group 4 includes electric chorea and Dubini's chorea.

CHOREA MINOR, SYDENHAM'S CHOREA OR ST. VITUS' DANCE, AND CHOREA INSANIENS

Taken collectively, this group may be regarded as a disease of the central nervous system, brain, medulla, and spinal cord, caused by the action of toxins, which give rise to mental and motor manifestations of an irritative nature, and which may run an acute, a subacute, or a chronic course, either with or without remissions.

Etiology.—The affections classified under this group occur most frequently between the ages of six and fourteen, although no age is exempt. The disease is frequently seen in children under six years of age, and also after the fourteenth year. Chorea insaniens occurs with special frequency in women during pregnancy. The proportion of females to males that are affected is about 3 to 1. A large majority of the individuals affected are found among the children of the classes who live amid unhygienic surroundings, who are poorly fed, and who dwell in poorly ventilated houses.

A nervous temperament, anemia, overwork, fright, trauma,

pregnancy, affections of the throat and nose, rheumatic conditions, etc., are among the frequent predisposing conditions. This disease sometimes follows the infectious diseases, such as scarlet fever, influenza, diphtheria, typhoid, etc.

The disease occurs more frequently in the early spring and early summer than at other periods of the year. In many cases one attack predisposes to another, and in a large number of cases relapses are prone to occur.

Pathology.—Pathologic examinations are rarely made in cases of acute chorea, but in practically all reported cases a dilatation of the capillaries in the nervous system was discovered. Punctiform hemorrhages, perivascular infiltration, softening, and granular degeneration of the cells in the gray matter of the cortex, in the basilar ganglia, medulla, and the cord were also seen, and in some places thickening of the meninges was present. These pathologic findings simulate the characteristic appearance of the nervous system in the chronic toxemias.

Upon taking into consideration the various causative factors of this disease, one is forced to the conclusion that there is no specific toxin, nor does the toxic substance always originate in a similar manner. Undoubtedly, a group of cases are seen in which the toxin is of bacterial origin.

In a large proportion of cases chorea develops along with or follows rheumatic manifestations, with or without true endocardial involvement. In these cases diseases of the nose and throat are commonly present. From a careful consideration of a number of cases as to development, course, and results of treatment, I am firmly convinced that certainly 40 per cent. of the acute choreas are caused by the absorption of toxic substances which are formed as the result of bacterial activity, and that organisms gain entrance into the system through the structures in the pharynx, the nose, or the nasopharynx.

Still another group of cases are seen in which local disease can be absolutely excluded, and which run their course without temperature; these cases follow fright, shock, and worry. It is my opinion that in this group of cases toxic substances form in neurotic individuals as the result of perversion of bodily metabolism. Chorea occurring during and after pregnancy may be due

to the formation of a toxic substance as the result of perverted metabolism, or may follow an infection.

In many cases a combination of these factors certainly exists, as where the individual lives under unfavorable hygienic conditions, has insufficient food, and is subject to mental stress or strain; in these cases toxins may be formed due to perversion of metabolism, and the general resistance of the individuals is lowered. This state predisposes to the entrance of pathologic organisms into the system.

Portions of the Nervous System Involved.—Many of the symptoms, such as restlessness, depression, irritability, weakness of memory, excitement, etc., are due to the action of a toxic substance on the cerebral cortex. The motor manifestations cannot be directly caused by the irritation of the cerebral cells, for the movements consist of irregular, uncontrollable, inimitable, unexpected, and purposeless contractions of muscles or groups of muscles. These movements are not characteristic of cortical activity, since the latter always brings about purposeful acts, whereas a consideration of the relation of the grouping of cells in the spinal cord, with the segmental connection of these groups to the various muscles of the extremities and trunk, makes it clear how irritation of the cells or groups of cells in the anterior horns can bring about the characteristic movements of chorea. Moreover, in many cases of chorea the reflexes are abolished or markedly decreased, and this could take place only as the result of spinal cord or peripheral nerve involvement. In many cases the respiratory rhythm is interfered with, peculiar movements occur about the face, strange sounds are made, speech is impaired, etc. These symptoms clearly point to involvement of the medulla. It would, therefore, appear to me that in chorea there is an involvement of the brain, medulla, and spinal cord.

Symptoms.—Abnormality of movement and mental peculiarities are the two principal manifestations of this disease.

Motor Symptoms.—At first more or less motor restlessness may be present, the individual being unable to remain still for any length of time; or the movements become awkward, and there is a tendency to drop things. Later on, or even from the onset, peculiar spasmodic movements occur; these movements

are unexpected and inimitable; they may be very slight, and only interfere with complicated acts, and not be noticeable when at rest, or they may be more marked, every movement involving fine coördination becoming impossible. These movements cannot be arrested by the will for any length of time, and are increased by attention and by excitement. The movements may be slight and of only momentary duration, but in some cases they become more or less general, and may be violent and continuous.

These abnormalities of movement more commonly affect the face and the extremities; in many cases irregularities in the respiratory rate can be noticed.

When the movements affect the upper extremities, abduction or adduction of the arm occurs, together with peculiar movements of the shoulder; the fingers are spread out, flexed, and extended. When the face is involved the forehead becomes wrinkled, the mouth drawn to one side, and the individual makes queer grimaces; the cheeks twitch, the movements of the tongue are irregular, unexpected movements of the eyes and eyelids occur, and the head is thrown from side to side. The trunk may be rotated. When the laryngeal and respiratory muscles are affected peculiar noises are made, or peculiar sounds may be uttered. Sometimes inability to swallow develops. The legs are also frequently affected, and walking is interfered with; the patient may stagger, or the gait become jerky and uncertain. It is only in very severe cases that walking becomes impossible. Weakness in the affected muscles may occasionally be marked, but complete or permanent paralysis does not occur.

The involuntary movements are not limited to one group of muscles, but occur at irregular intervals in the different groups. Both the distribution and the occurrence are characterized by this irregularity—the movements which are purposeless constantly vary in form and in direction. Awkwardness of movement is always present.

There is practically no disturbance of sensation. The tendon-reflexes may be normal, increased, or absent; ataxia may occur, and in some cases is a very prominent symptom. One side of the body may be affected to a much greater extent than the

opposite side, and in some instances the symptoms are limited practically to one side of the body, this condition being known as hemichorea. In many of the more severe cases difficulty in talking develops and speech sometimes becomes impossible. In severe cases more or less constant violent movements take place, and the individual is never still; he may frequently injure himself by coming in contact with furniture or other objects, and great exhaustion results.

As a rule, the movements cease during sleep and are increased by voluntary movement.

Mental Symptoms.—These patients are usually very emotional, laughing or weeping being easily provoked. Earlier in the disease there is marked irritability, and this often causes the individual to be regarded as disagreeable and cross until the nature of the malady is discovered. The patient's disposition generally undergoes a change, and he becomes cross, quarrelsome, depressed, and unable to exercise self-control. In some cases the mind seems to become weak. Emotional excitement usually increases the movements. In many cases there is difficulty in fixing the attention, and disturbance of memory is also present.

In adults, but rarely in children, there may develop a marked depression or delirium, sometimes accompanied by acute maniacal excitement, confusion, and hallucinations or delusions. This marked mental state generally develops at the height of the disease, giving rise to what is known as chorea insaniens. The most severe types occur during pregnancy, in the puerperal state, and in general infections.

Paralytic Chorea.—There is one type of chorea in which, instead of the marked abnormality of movement, an apparent paresis of a part or of the entire body occurs. It is first noticed that the individual uses one side of the body less frequently than the other, or avoids using one side altogether. This motor involvement extends and may involve the entire body. The movements of the limbs or body may still be performed, but only in a very feeble way; the muscles become flaccid, and the tendon reflexes are decreased or abolished. Transient tingling develops, which becomes more marked during the course of the disease. When

the pseudoparalysis affects the muscles of articulation, mutism develops. This form of chorea is known as paralytic chorea. Its course differs from the ordinary type of chorea only in the onset, choreic movements developing during the course of the disease, revealing its true nature. Muscular atrophy does not occur.

The general condition of the patient depends to a great extent upon the severity of the movements, the amount of rest the patient gets, his ability to take nourishment, and upon the complications which may be present. Among the last may be mentioned endocarditis, articular rheumatism, pyrexia, etc. Individuals suffering with chorea are usually pale and badly nourished, have little appetite, and are constipated. In about 40 per cent. of cases heart murmurs are present; these may be functional, hemic, or due to endocarditis. Fever and acceleration of the pulse-rate are present in many cases.

Course.—The onset of this disease is usually gradual, but in some cases it may appear suddenly, as after fright or following mental strain. In mild cases the movements may be so slight as to be detected only upon careful examination, the mental and emotional symptoms being more prominent; in other cases there is a gradual increase in the motor symptoms, which may become so severe or so constant as to interfere with the patient's rest or with his ability to take nourishment. In some cases the movements are so violent that the patient must be surrounded by mattresses or similar soft articles in order to prevent him from severely injuring himself. In most cases the mental symptoms never reach an alarming degree of severity, but when the chorea develops in adults, and especially in pregnant women or in neurotic subjects, or if it follows a severe infection, marked mental symptoms may occur. In the milder cases the usual duration is about five weeks; the severer cases last from five weeks to a year, and some cases last for a very prolonged period. In the majority of cases occurring in children the duration is about ten weeks, the onset being gradual, and the condition subsiding gradually.

Prognosis.—The prognosis as to life and as to recovery is in nearly all cases good. Death occurs only in the exceedingly

severe forms, in which muscular contractions are so severe as to make sleep and nutrition impossible; in these cases death is due to exhaustion or to cardiac degeneration. Death may also occur as the result of injuries. Organic cardiac affections occurring in very young individuals do not seem to affect the prognosis. When this condition exists in older individuals the prognosis as to life is more serious.

Rapid emaciation, delirium, and a rise of temperature occurring during the course of chorea are serious signs. Chorea occurring during pregnancy or cases in which there are marked mental manifestations present a more serious prognosis. In adults a form of chorea may occur that runs a chronic course and lasts for years or during the entire lifetime.

Chorea shows a marked tendency to recur, the intervals between attacks usually lasting about a year. In some cases, however, attacks may follow one another in quick succession; in others several years may intervene between attacks. Generally not more than one or two relapses occur, but a greater number have been observed. The older the patient, the more likely is the disease to run a protracted course, but this does not preclude the hope of recovery.

Diagnosis.—The diagnosis of this disease is usually very easily made. Some difficulty may be experienced in distinguishing a chorea that begins with weakness from a *paralytic condition*, but a careful study of the case will reveal the muscular twitchings and the characteristic mental state which will enable one to reach a correct diagnosis.

Chorea may be mistaken for *general tic*, or in those cases in which the choreic movements are limited to certain parts of the body or to the face it may be mistaken for localized tic; the differentiation is made from the fact that the movements in tic are purposive and can be reproduced voluntarily, whereas in chorea the movements are purposeless and cannot be imitated; moreover, in tic the mental state is normal, whereas in chorea the individual is emotional and irritable. Temperature, pulse, and cardiac disturbances are frequently present in chorea, whereas they are generally absent in tic.

Chorea occurring in adults must be differentiated from Hun-

tington's chorea, senile chorea, chorea accompanying organic brain lesions, etc. From *Huntington's chorea* it is differentiated by the history of the case and by a consideration of the progress of the disease: in Huntington's chorea the disease usually manifests itself for the first time at about the fortieth year, the abnormal movements commonly beginning in the hands, and gradually extending until the entire body is affected; the mental symptoms develop gradually and finally lead to dementia. From *senile chorea* the condition is differentiated by the age of the patient, by the prominence of the mental disturbances, and by the fact that the choreic movements are usually accompanied by more or less generalized tremors. From *chorea accompanying organic cerebral disease* this condition is differentiated by the history of the symptoms following some organic neural disease, by the presence of the symptoms of that disease, and by the fact that the choreic movements are usually limited to the portions of the body that are under control of that portion of the central nervous system that is organically affected.

Treatment.—The patient's room should be light, well ventilated, and quiet. Older children and adults should be confined to bed, and for younger children as much rest as possible should be insisted upon. The patient should be isolated, only one or two persons being in attendance. The diet should be light and nutritious, and the patient should be encouraged to drink an abundance of water. The bowels should be kept well open, and a daily warm bath is to be given. Gentle massage is often beneficial. In those cases in which the movements are at all violent or severe the patient should be kept in a bed that is well padded and be carefully watched day and night so that he does not injure himself. In the case of adults in whom marked mental symptoms develop it may be necessary to remove the patient to an institution so that he may be cared for properly.

The cause of the condition should be carefully sought, and if found it should be treated and, if possible, removed. In those cases in which diseased conditions of the throat and nose exist proper medical and surgical treatment should be instituted. It has been my experience that in many cases proper treatment directed toward the throat and nose has tended to shorten the

duration of the disease, and has cured cases that had existed with slight remissions for a number of years; such treatment has also tended to prevent relapses and recurrences.

When chorea occurs in pregnant women, it may be necessary to terminate the pregnancy in order to save the patient's life; this measure should be resorted to only when there is a condition of extreme exhaustion, or in the presence of organic heart disease, marked mental manifestations, etc. In all cases the bowels should be freely opened by the administration of calomel, followed by castor oil or a saline purgative. In those cases in which a rheumatic tendency is present or in which a rise of temperature and cardiac involvement occur, sodium salicylate, aspirin, salicin, etc., in doses oppropriate to the age, should be given every three or four hours. Antiseptic nose and throat sprays should also be used. Patients who are pale and debilitated should receive arsenic, preferably in the form of Fowler's solution; this should be given three times a day in increasing doses, beginning with three drops and increasing the dose until the physiologic limit of the arsenic is reached, which is manifested by puffiness of the eyelids, etc. Arsenous acid, combined with iron, or subcutaneous injections of cacodylic acid, in doses of from $\frac{1}{3}$ to $\frac{2}{3}$ grain, may also be used.

If the arsenic cannot be taken without discomfort, or if it gives rise to disagreeable symptoms, its use should be discontinued, and in its stead the iron preparations may be administered; for children the syrup of the iodid of iron, in doses ranging from 10 to 30 drops three times a day, is very efficacious.

If malarial infection is suspected, quinin should be given. When the movements are extensive and persistent, or when restlessness is marked, the bromids exert a soothing effect; in some cases in which these fail better results are obtained from chloral or from a combination of bromid and chloral. In very severe cases it may be necessary to use chloroform. Veronal, medinal, adalene, etc., are all of service in inducing sleep.

Cardiac involvement, which is one of the most frequent complications, is best treated by rest in bed, stimulation, if necessary, with caffein, and the application of ice-caps to the precordial region.

For a considerable period after the attack these patients should avoid excitement and overexertion. Tonics and, if possible, a change of climate for a time should be ordered.

HUNTINGTON'S CHOREA (CHRONIC PROGRESSIVE HEREDITARY CHOREA)

Etiology.—This is a somewhat uncommon disease, which is transmittied from one generation to another. It was first described by Dr. Huntington, of East Hampton, Long Island. The disease may skip a generation, or other forms of nervous disease, such as epilepsy, hysteria, etc., may occur in its stead. Males are affected somewhat more frequently than females. The disease generally appears between the ages of twenty and forty, but may develop earlier or later. When it is transmitted through several generations it shows a tendency to develop later in life. In some cases the disease appears gradually without an exciting cause; in others the initial symptoms follow shock, injury, emotional disturbance, pregnancy, etc.

Pathology.—Changes of a sclerotic nature affecting the meninges, disseminated chronic inflammatory areas in the cortex of the motor region, changes in the wall of the blood-vessels, and sclerotic patches in the basal ganglia have all been described in this condition.

Symptoms.—A gradual restlessness and peculiarity of movements, at first limited to the upper extremities, the face, and the neck, develop. These symptoms gradually increase in severity and in extent, until all voluntary movements of the body are affected. These movements resemble those of chorea minor; they are irregular and more or less constant. The peculiar contractions which involve muscles or groups of muscles bring about strange, inimitable, and uncontrollable movements of the extremities, trunk, face, neck, etc. The ocular movements are not usually affected. The gait is uncertain and jerky, and at times the individual is thrown to the ground. Speech is interfered with by twitching of the tongue, and by the movements of the lips and the spasmodic respiratory efforts. The

muscles of the face contract, giving rise to grimaces. All these movements can be suppressed for a time by an effort of the will, and they generally cease during sleep. They become more intense and violent as the result of mental or physical exhaustion.

Fig. 155.—Huntington's chorea. Senile chorea.

The motor power remains practically normal during the entire course of the illness. Hemiplegia may develop in the later stages of the disease. There are no disturbances of sensation, but the reflexes are usually slightly increased. The internal organs are not affected (Fig. 155).

The movements, which are always bilateral, generally cease

during sleep. Gradually, during the course of the disease, the second characteristic symptom develops; *i. e.*, a gradual progressive mental feebleness. These patients may be very irritable from the onset, or, on the other hand, a state of indifference may be present. Weakness of memory and inability to fix the attention gradually appear, the judgment becomes defective, and the patient becomes very emotional. In some cases marked psychosis appears, the patient developing delusions or hallucinations, or he becomes excited or depressed. Occasionally attempts at suicide are made.

As the disease progresses the mental weakness increases, and a gradual weakness and rigidity in the legs and arms develop. Most cases must be committed sooner or later to an institution.

Course.—The course is gradually progressive, the condition persisting for many years.

Prognosis.—The prognosis is very unfavorable, the disease being practically incurable. The individual eventually succumbs to some intercurrent disease or as the result of interference with nutrition, etc.

Diagnosis.—The diagnosis is made from the hereditary nature of the condition, and from the fact that it develops usually between the thirtieth and the fortieth year; also by the development of mental symptoms during the course of the disease.

It is differentiated from *senile chorea* by the fact that in this condition no hereditary influence can be traced, and, moreover, the disease develops later in life.

Treatment is purely symptomatic. Drugs do not seem capable of arresting the progress of the disease. The patient should be carefully nourished, and when his mental condition becomes so disturbed that it is no longer possible to care for him properly in the home, it is advisable to remove him to an institution properly equipped for the care of the insane.

CHRONIC PROGRESSIVE CHOREA OF ADULTS AND SENILE CHOREA

These forms of chorea usually occur between the ages of fifty-five and eighty. Males are affected slightly more fre-

quently than females. A history of a neuropathic taint in the family is frequently obtained.

Pathology.—The pathologic changes that take place in this condition are somewhat similar to those described in Huntington's chorea. There is no satisfactory explanation of the manner in which these changes are brought about.

Symptoms.—The abnormal movements resemble those that occur in other types of chorea. The muscles of the upper extremities, neck, face, and tongue are those most frequently affected. Articulation and swallowing may be greatly interfered with. A moderate degree of muscular weakness may be present.

These patients are very emotional and irritable, and in some cases marked mental weakness develops. Groups of cases are seen in which the choreic movements and the mental weakness are progressive. These patients are distinguished from those suffering from Huntington's chorea only by the absence of the hereditary history.

Course.—In these cases, as in the ordinary types of chorea, the movements are increased by emotional disturbances, and usually cease during sleep. They may appear more or less suddenly after shock, anxiety, injury, or exhausting conditions. They usually run a slowly progressive course.

Prognosis.—The abnormal movements may continue for prolonged periods, persisting until death takes place, which may not be hastened by the presence of this condition. Except in a very few instances recovery is not to be looked for.

Diagnosis.—The diagnosis is made from the presence of the characteristic movements, which cease during sleep and are rendered worse by emotional excitement and on observation.

Chronic progressive chorea of adults is distinguished from Huntington's chorea by the absence of a hereditary history. The diagnosis of senile chorea is made from the age of the patient and the usual presence of tremor.

Cardiac affections, when they occur, are generally of a degenerative nature.

The **treatment** is the same as that prescribed in chorea minor, and includes rest, general building up of the system, antirheumatic remedies, arsenic, tonics, and sedatives, if necessary.

CHOREA ACCOMPANYING ORGANIC BRAIN LESIONS

Choreiform movements may develop during the course of or follow organic cerebrospinal disease. When they occur, these abnormal movements differ in no way from those seen in chorea minor. The organic diseases of the nervous system that are most frequently followed by choreiform movements are hemiplegia, meningitis, idiocy, etc.

In posthemiplegic chorea the movements are generally limited to the affected side, but they may be bilateral. It is, of course, possible for an acute chorea to develop in a mentally deficient individual, in one who has had meningitis, or in one who has sustained a hemiplegic attack; when it occurs under these conditions the chorea differs in no way from the ordinary form of chorea, except that the outlook for recovery is not quite so good. If, on the other hand, the choreiform movements follow the organic disease more or less closely, the outlook as regards cure of the choreiform movements is extremely unfavorable, the condition generally becoming permanent.

In these patients emotional disturbances tend to increase the movements, whereas they generally cease during sleep. Athetoid movements are also frequently present in those individuals.

The **treatment** should be directed toward the underlying organic disease. If the restlessness is marked, it may be necessary to quiet the patient by the administration of bromids and chloral.

CONGENITAL CHOREA

Choreiform movements may be present at birth or appear shortly after. The children in whom these abnormal movements occur, if they live, are apt to be idiots, imbeciles, or very slow in their intellectual development.

In addition to the choreic movements and mental deficiency there is generally present a weakness or a paralysis which may be of the flaccid or spastic type; athetoid movements are also usually present.

As a rule, the movements affect the face or the upper extremities to a greater extent than the trunk and the lower extremities.

They are similar to the movements of chorea minor, except that they are apt to have a greater range. They resemble the movements of chorea minor in so far as they are rendered worse by emotion and are generally suspended during sleep. No relationship exists between this form of chorea and rheumatism—cardiac lesions are not present, nor is there any increase in temperature, joint involvement, etc.

The course of the disease is chronic, and treatment has no effect upon the condition.

EPIDEMIC CHOREA

Epidemic chorea was first known to occur in Germany during the fourteenth and fifteenth century. It is completely distinct from the forms of chorea that have previously been described. The movements may have nothing in common with true choreiform movements, but as the result of imitating others peculiar movements of the extremities and body occur, and the individual jumps, dances, etc. Epidemics of this kind occurred as early as the thirteenth century, and have been described from time to time at later periods.

Etiology.—These epidemics occur only under unusual conditions, as when groups of persons are laboring under strong emotional excitement or are subject to intense nervous strain. It is under such conditions that imitation is especially apt to occur, the individual being generally in a receptive mood and calm judgment being quite impossible.

Symptoms.—An individual or a group of persons imitate or originate movements which are purposive in character and can be produced voluntarily. In some instances the movements become intense and violent, and continue until the individual falls from sheer exhaustion.

Course.—The onset is usually more or less sudden, and the condition may last a longer or a shorter period. Exacerbations or remissions occur, and there is either a sudden or a gradual termination, the former being most frequent. The epidemics are usually of gradual growth and recede slowly.

The **prognosis** is good, and the **treatment** is the same as that applied in hysteria.

ELECTRIC CHOREA

Various conditions have been described under this term.

Dubini's disease is a form of electric chorea seen most commonly in the northern part of Italy, but even there it is of very rare occurrence. It is believed to be an acute infection, in which toxic substances first attack the neurons of the cerebral cortex. This is manifested by the frequent onset of the disease with convulsions, and by the occurrence of unilateral symptoms, which later become generalized. As the disease progresses the anterior horn cells become affected, and muscular atrophy occurs.

Symptoms.—The disease usually begins with pain in the neck and back and fever, which varies in degree according to the severity of the affection. Generalized or convulsions limited to one side may occur. In conjunction with the convulsions, or even in their absence, the characteristic features of the disease appear—rigid muscular contractions, similar to those elicited by electric stimulation. First one arm and one side of the face is involved, and later the leg upon the same side; finally the opposite side of the trunk becomes affected. If the condition progresses, paralysis of an atrophic type occurs, the portions of the body becoming affected in the same order in which the muscular contractions occur. The weakness is gradually progressive until, in the end, it becomes general. Atrophy with reaction of degeneration takes place. The disease runs a progressive course, death occurring in from a few days to several months.

Under the term electric chorea Bergeron has described an affection which occurs in anemic children, and is usually observed between the ages of seven and fourteen.

Etiology.—This form of electric chorea is believed to be caused by some form of gastric irritation.

Symptoms.—In this condition violent twitchings occur which come on in spasmodic attacks. The movements are rhythmic and are similar to those which can be produced by regularly interrupted electric stimulation. These movements are usually

limited to certain groups of muscles, and are purposive in character; they also resemble movements which could be produced voluntarily: the head may be tossed from side to side or backward and forward; the shoulders may be elevated, the forearms flexed and extended, etc. These movements may be confined to one extremity, to single muscles, or may involve the entire musculature of the body. They are rendered worse by emotional excitement and by attempts to repress them, and disappear during sleep. Examination of the patient for pathologic conditions is negative.

The **prognosis** in these cases is very good. Usually after running a very brief course the disease ends in complete recovery.

Diagnosis.—This condition is to be differentiated from hysteria, such differentiation being difficult and often impossible. The limitation of the movements to certain parts of the body and the constancy of involvement of that particular part of the body should assist one in making the differentiation.

Treatment.—The affected child should be isolated, and placed amid pleasant hygienic surroundings. It is most important that the gastro-intestinal tract be properly cleansed by the administration of calomel, castor oil, or other purgatives. The diet should be regulated, and should be of a light, nutritious nature. As these children are usually anemic and undernourished, tonics, and especially arsenic, are indicated. Cold douches, Scotch douches, and, as soon as possible, proper gymnastic exercises are all to be recommended.

Henoch has also described a form of electric chorea which occurs in childhood. This condition is also characterized by lightning-like muscular contractions which affect especially the muscles of the neck and shoulders. The affection described by Henoch closely resembles the type of chorea described by Bergeron.

PARALYSIS AGITANS (SHAKING PALSY; PARKINSON'S DISEASE)

Paralysis agitans is a disease of the nervous and muscular system, which manifests itself usually between the fortieth and

the sixtieth year. It is characterized by the occurrence of tremor, muscle rigidity, and abnormal sensations.

Etiology.—This disease, as stated, manifests itself generally between the fortieth and the sixtieth year, although it occurs in rare cases before the thirtieth year and in a considerable number of cases after the sixtieth year. It is more common in men than in women. Heredity may play a small part in acting as a predisposing cause, but it is certainly not of much importance. In many cases no ascertainable cause can be discovered. The most common and most important exciting causes of this condition seem to be prolonged anxiety, worry, emotional shock, fright, overwork, and exposure. In some cases the disease sets in within a short period after exposure or injury, or after prolonged worry and anxiety.

Pathology.—The lesions which are most commonly found in paralysis agitans are degenerative changes in the vessels and a proliferation of the glial tissue; the muscle-fibers also show slight atrophic changes; some of these changes are certainly of a senile nature; they are not found constantly in all cases. At the present time there is no consensus of opinion as regards the cause or pathology of the disease, and it is considered to be a functional condition.

It is probable that with other methods of investigation a true causative factor and most likely pathologic changes will be found.

Symptoms.—The disease is generally of slow onset, although in a few cases the symptoms rapidly follow fright, exposure, or marked emotional disturbances. In cases of gradual onset, usually for some considerable time, the patient complains of an aching, stiff feeling in the back and extremities, and later the characteristic symptoms of the disease gradually develop. The most common symptom is tremor, which affects chiefly the extremities, especially the upper, and particularly the hands and fingers. The tremor usually affects one extremity first, and early in the disease one limb is involved to a greater extent than the other. As the disease advances the limbs become equally affected.

The tremor is characterized by the fact that it occurs while

the patient is at rest, and that, early in the disease, it can be inhibited by an effort of the will. It is of a slow type, four or five oscillations occurring to the second.

The range of the movements is usually within narrow limits. In the upper extremities the tremor takes the form of flexion and extension and abduction and adduction of the fingers and flexion and extension and pronation and supination of the hands. The tremor of the thumb and index-finger is of such a character as to give rise to a movement as if one were rolling a pill. After the disease is once well extablished, the tremor is more or less constant in amplitude and in rate, although it may be inhibited temporarily by volition or during the performance of some purposive act. It persists, nevertheless, or is sometimes increased while making continuous movements, such as in writing, etc. The tremor is practically always aggravated by excitement and by emotional disturbances. The hand-writing of the patient is characterized by the smallness of the letters and by the symmetrically wavy outlines and strokes.

When the tremor affects the lower extremities, extension, flexion, adduction, and abduction of the foot usually occur. The muscles of the thigh are also often involved. The tremor does not, as a rule, involve the muscles of the neck, but not uncommonly it affects the muscles of the lower jaw, chin, lips, and tongue. The tremor generally ceases during sleep.

The second of the cardinal symptoms is chronic muscular rigidity, which affects practically the entire musculature of the body. The muscles of the back, neck, and throat are usually affected to a more marked extent, and in the great majority of cases the muscles of the face are involved. This muscular rigidity causes certain changes of posture. The muscular rigidity and its subsequent alterations of posture may constitute the first sign of the disease. The attitude of a patient in whom the muscular rigidity is at all marked is one in which the head and trunk are bent forward, the arms held close to the side, the elbows slightly flexed, the wrists slightly extended, and the fingers partially flexed at the metacarpophalangeal joints, whereas the middle and terminal phalanges are extended, with the thumb adducted (Fig. 156). The legs are moderately flexed at the hip- and

knee-joints, with often an adduction of the thighs. As the result of rigidity of the facial muscles the face presents a peculiar, expressionless, mask-like appearance. Even early in the disease a slowness or clumsiness of complex voluntary movements are noticed; it appears as if there were a delay in the conversion of a volitional impulse into a muscle response, and as if the muscles responded more slowly than normally. This is well seen in the difficulty the patient experiences in dressing and in undressing

Fig. 156.—Position of hands in Parkinson's disease.

and in changing his position. Many of these patients must be assisted in dressing and in undressing, and later in the disease in changing their position. The gait also undergoes a deviation from the normal. The patient stoops forward, walking slowly at first, and then gradually more and more rapidly. He appears as if about to fall forward—a condition that is called propulsion (Fig. 157). If these patients are told to walk backward, sometimes the same condition occurs, and they either fall or

their progress is stopped by some immovable object; this condition is termed "retropulsion."

The tendon reflexes are usually increased, but may be normal. True clonus and the Babinski sign are never present. Paralysis does not occur, and even in the most advanced cases some degree of motility is retained; this may, however, as the result

Fig. 157.—Posture in Parkinson's disease.

of the muscle rigidity and of retardation of muscle response be very limited.

Speech is usually very monotonous. The voice is generally low, and at times the individual has difficulty in producing the first sound, but as soon as this is done the words flow rapidly, and in some cases more rapidly than normal. Hyperidrosis and an increased flow of saliva occur very commonly, and in

some cases salivation appears, giving rise to considerable discomfort.

Sensation.—Tactile, pain, temperature, and muscle sensations are not disturbed. Pain, which is subacute in character and described as an aching and stiffness, is generally complained of from the first; in fact, many of these patients are treated for rheumatism long before the true condition is discovered. In many cases the sense of stiffness and aching is complained of throughout the entire course of the disease.

Paresthesias, such as a sensation as of heat—a feeling as if hot water were being poured over the body; or a sensation of cold—a feeling as if cold air were being blown upon the body, numbness, tingling, etc., are very frequently complained of. These paresthesias are among the most constant symptoms of the disease.

The mental state of the patient is usually unimpaired, but as the disease progresses these patients become anxious, restless, irritable, depressed, and emotional; in some few cases the opposite condition develops, and in spite of the physical handicap the patient develops a certain geniality of disposition.

Course.—The development and progress of the disease are usually slow. It may begin with tremor or with a sensation of aching and stiffness—first in one extremity, and then gradually spreading. In some cases it appears suddenly after fright or emotional disturbance. The posture, gait, mask-like expression of the face, etc., gradually become manifest as the disease advances. At any time during the course of the disease remissions may occur. It usually runs a gradually progressive course, lasting many years.

Prognosis.—The prognosis as to recovery is very unfavorable; as to life it is good, for the affected individuals do not die of this disease. Hemiplegic and apoplectic attacks may occur during the course of the disease, but are no more prone to occur in these patients than they are in unaffected individuals of the same age.

Diagnosis.—When the disease is once well developed, little or no difficulty will be experienced in making the diagnosis. The appearance of the characteristic tremor, the aching, and the muscular rigidity easily lead one to a correct conclusion. The

disease is differentiated from *multiple sclerosis* by the fact that in paralysis agitans the tremor is decreased upon voluntary effort and increased in multiple sclerosis. In the former disease the tremor is of limited amplitude, and in the latter disease the amplitude increases upon voluntary effort; in the former disease nystagmus does not occur, whereas in the latter it is frequently present. The speech in the former disease is weak, whereas in the latter it is slow and scanning. Paralysis agitans is a disease of advanced life, whereas multiple sclerosis occurs in young adults.

Paralysis agitans must sometimes be differentiated from *paresis:* in the latter disease the tremor is finer, is increased upon voluntary motion, and pupillary disturbances are present, as well as disturbances of speech, mental manifestations, etc., all these symptoms being absent or quite different in paralysis agitans. From *senile tremors* paralysis agitans is differentiated by the fact that in senility the tremor generally involves the head, the legs being rarely involved; there is an absence of the mask-like expression of the face, the posture of the hands and body is quite different from that of paralysis agitans, and in advanced cases paralysis, sensory disturbances, etc., are frequently present.

Treatment.—A person afflicted with paralysis agitans should lead a life free from excitement and worry; he should live amid pleasant surroundings, and if his condition permits, he should follow a congenial occupation. Change of scene is also advisable.

Overexertion, both physical and mental, should be absolutely avoided, since it hastens the progress and intensifies the symptoms of the disease. All strenuous treatment, such as violent exercise, very hot or very cold baths, deep-seated massage, and the like, is contraindicated. Warm salt rubs, sponging with warm water, and gentle massage are of benefit, and one of these measures should be carried out at least three times a week. The diet should be light and nutritious, and the bowels be kept freely open. Exposure in too cold or too hot weather is to be avoided.

At the present time no drug is known that has a curative effect

upon this disease. There are several remedies, however, which tend to render the patient much more comfortable and to a certain extent to decrease the tremor; such remedies, even when administered over a period of several years, do not seem to produce harmful effects. Tincture of veratrum viride, given in doses of from 3 to 5 drops three times a day, will sometimes lessen the intensity of the tremor; tincture of gelsemium also at times exerts a beneficial effect. Sulphate of duboisin is also highly recommended; hyoscyamin hydrobromid, $\frac{1}{100}$ grain three times a day, has given very good results, especially in those cases in which there is an increase in the salivary secretions.

I have found that the combination of codein with these drugs usually tends to increase the comfort of the patient, particularly when there is much complaint of muscle stiffness.

EPILEPSY (FALLING SICKNESS)

Epilepsy is a disease characterized by a sudden disturbance of consciousness, which may occur with or without warning, and which may be attended by more or less marked disturbance of motility. Three forms of epilepsy are known—major epilepsy, minor epilepsy, and psychic epilepsy. Major epilepsy, or *grand mal*, is a condition in which a sudden loss of consciousness occurs with or without warning, which is immediately followed by a generalized convulsive seizure which is at first tonic and then clonic.

Minor epilepsy, or *petit mal*, is a condition characterized by sudden loss of consciousness, which is usually of very short duration, and which may or may not be attended by abnormalities of movement, which, if present, are generally of very moderate degree.

Psychic epilepsy is a condition in which, either with or without warning, there is a sudden perversion of consciousness, which may endure for a long or a short period, during which the individual is capable of carrying on complex activities of which he has absolutely no recollection after the attack disappears.

Epileptiform seizures may be either idiopathic,—*i. e.*, occur without ascertainable cause,—or they may be symptomatic,—

that is, they may be the evidence of a disturbance or a perversion of the functions of the brain due to some underlying disease.

Jacksonian epilepsy is a type of symptomatic epilepsy in which there is no disturbance of consciousness. It is caused by irritation of a localized area of the brain.

Nocturnal epilepsy is a term applied to epileptic seizures that occur only during the night, and may remain undetected for a long time.

Etiology.—*Heredity.*—In a large number of cases of idiopathic epilepsy there is a distinct hereditary history of the existence of a neuropathic taint, debilitating disease, mental disorders, syphilis, chronic intoxication, especially alcohol, etc. In a small proportion of cases there is a history of epilepsy having existed in previous generations.

Age.—In a great many cases epileptic attacks appear for the first time before the age of twenty. In about one-third of the cases they appear before the tenth year, and in another third before the twentieth year. Epileptiform seizures which occur for the first time after the twentieth year should always arouse grave suspicion of the presence of an organic disease, or of a toxic condition of exogenous or endogenous origin. The disease occurs somewhat more frequently in males than in females.

Chronic poisoning from alcohol or lead, or toxic conditions of the gastro-intestinal tract may either bring about changes in the cerebral substance and predispose to the occurrence of epileptic seizures, or, as the result of an acute poisoning, may be the direct exciting cause of an epileptiform seizure. Acute infectious diseases, such as scarlet fever, measles, typhoid, influenza, etc., may be followed by epilepsy; it is probably under these conditions that organic changes have taken place in the cerebral cortex, as the result either of a toxemia or of an infection.

Trauma.—Injury to the head may cause localized cortical lesions that are followed by Jacksonian or general epileptiform seizures. Blows or falls, without apparent localized cerebral lesions, may be the exciting cause of generalized epileptiform seizures. Syphilis may be the etiologic factor in generalized epileptiform seizures, but in most cases the epileptiform seizures that occur in cerebral syphilis are merely symptomatic of the

underlying disease. Prolonged exposure to very hot or very cold weather, overexertion, fright, anxiety, long-continued worry, etc., are also among the probable exciting causes of the disease.

Reflex Causes.—In predisposed individuals a prolonged nerve irritant is capable of precipitating an epileptic condition. Among the common reflex causes are errors of refraction, intestinal worms, diseases of the throat and nose, caries or unerupted teeth, phimosis, diseases of the heart, abdominal organs, or pelvic viscera, and scars pressing upon or including nerves, etc.

Chronic Cerebral Disease.—Meningitis, hydrocephalus, vascular disturbances, anomalies of development, etc., may also be the underlying causes of epilepsy.

Chronic nutritional disorders, such as rickets, scurvy, disturbance of the secretions of the ductless glands, nephritis, diabetes, tuberculosis, etc., may be predisposing or exciting causes of epilepsy.

Pathology.—As a rule, a careful examination of the nervous system and of the organs of the body after death yields negative results. The findings which are discovered are certainly not constant. In many cases a careful histologic investigation has revealed the presence of atrophy of the nerve-cells, with proliferation of the glial fibers in the cortex.

The consensus of opinion at the present time is that in epilepsy the pathologic lesion is situated in the cortex of the brain. Several theories as to the causation of the attacks exist. Underlying all these theories is the assumption that a neural tissue in a state of easily disturbed equilibrium exists. When one takes into consideration the facts that epileptiform seizures can be produced by intentional irritation of the cerebral cortex; that they occur with such frequency in organic diseases of the brain, and that they occur so often in individuals who show evidence of maldevelopment of the brain; that when an aura exists, it is always constant and points to some local cerebral area; that individuals may show definite mental deficiencies without apparent histologic changes in the brain—when one considers all these facts one is led, more or less definitely, to the conclusion that the underlying cause of epilepsy is some deficiency in the

inhibitive power of the cortical cells; that this inhibitive power may be decreased either as the result of defective heredity or in consequence of organic destructive disease of the cortex; such destructive condition may be of such character as to be easily discernible, or, with the present methods of investigations, may elude discovery. Furthermore, that the inhibitory power of the cortical cells may be decreased, either slowly, by the prolonged action of the toxic substances, or more or less acutely, by being suddenly overwhelmed by large doses of poisonous substances; as the result of disturbances of nutrition of the cerebral cells, or because of the vascular disturbance where there is defective nourishment of these cells. The epileptic attacks are the result of some added irritant acting upon cells whose inhibitory power is below normal. These added irritants may be reflex excitations, emotional disturbances, toxemias, etc.

Symptoms.—In some cases the epileptic attack is the only symptom of the disease, the individual being apparently normal in the interval between the attacks. In about 50 or 60 per cent. of cases the epileptic attack is preceded by a warning, termed an aura, which occurs from a few seconds to some considerable time before the attack, and is always constant in character. In some cases, especially after the disease has persisted for a considerable length of time, symptoms of disturbance of the general health or changes in character, emotional tone, or mental condition appear in the intervals between the attacks.

The *aura*, or warning, which may be motor, sensory, vasomotor, or psychic, may not give the patient sufficient time, before the onset of the attack, to summon assistance or to take measures to protect himself from the consequences of the attack. In many cases there is an appreciable interval between the aura and the attack, and occasionally the aura may occur several hours or more before the onset of the seizure. The aura manifests itself in many ways, but is always constant in the same individual. The commoner types of aura are: Peculiarities of sensation, many patients complaining of a peculiar sinking, disturbed, empty feeling in the epigastric region; some experience a feeling of vertigo, numbness in the upper and lower extremities, peculiar mental states, such as a sense of fear, of

elation, of uncertainty, of restlessness, of depression, etc.; others have a peculiar sensation about the heart or in the abdominal cavity, nausea, a feeling of fulness in the head, sensations of heat or cold, or flushing, drowsiness, or excitement, hallucinations of sight, hearing, smell, and taste, sudden flashes of light or blindness, general trembling, increased secretion of saliva, attacks of diarrhea without cause, etc. Occasionally the aura may occur and no seizure follow.

The *attack*, which is the chief manifestation of the disease, occurs more or less suddenly, either with or without the aura. In the *grand mal* type of epilepsy there is loss of consciousness, in some cases accompanied by a sudden cry or groan; in other cases the cry may be absent and the head be drawn to one side or drawn backward, the eyes rotating either upward or downward; the pupils are dilated, the patient falls, and the face is pale; nearly all the muscles become tonically contracted, and the face gradually becomes cyanotic; it is during this state, which may last from a few seconds to from forty to fifty seconds, that the patient bites the tongue, owing to the fact that, in the tonic spasm, the tongue is protruded between the teeth, which close on account of the tonic contraction of the jaw muscles. The cyanosis is due to the tonic contraction of the thorax and abdominal muscles, which prevents respiration from taking place. It is also during this stage that the thumb is strongly flexed against the palm and the fingers flexed over the thumb; the arms are usually flexed, and the legs and feet are extended. The contents of the bladder and rectum are sometimes expelled as the result of the tonic spasms of the muscles that govern these organs.

Following this stage clonic spasms, at first limited in extent, but gradually becoming more violent in character, occur. These clonic spasms may affect all the muscles of the face, trunk, and extremities. During this stage the breathing becomes loud and stertorous, and saliva, sometimes blood-stained if the patient has bitten himself, flows from the mouth. At this time the patient may also bite his tongue or otherwise severely injure himself. The extent of the muscular movements varies in different patients, and depends upon the severity of the seizure:

in some individuals the convulsive movements are limited to a few muscles, and are only moderately severe, whereas in others the convulsive movements involve all the muscles and are extremely severe, and consist of violent thrashing movements of the extremities and trunk. Dislocations and fractures may occur during this stage as the result of the violent muscular movements.

The clonic stage lasts for from one to five or ten minutes, and sometimes longer. The muscular movements gradually become less severe, and the patient slowly regains consciousness. After the seizure the patient is dazed, does not realize what has happened, but gradually, within half an hour, he comes to himself. In many cases the patient passes from the convulsive state into one of deep sleep, which lasts for several hours, and from which it is difficult or almost impossible to arouse him. Other patients, after a brief period of dulness or confusion, become perfectly normal and are able to continue the occupation in which they were engaged when the seizure occurred.

Following an attack the patient is usually much fatigued, complains of weakness and headache, or of pain the result of an injury sustained during the attack. Generally, the last thing the patient remembers is the aura, or any duty with which he was occupied immediately preceding the attack. Patients can usually tell that they have had an attack from the feeling of exhaustion, weakness, and fatigue, or from the presence of an injury which was inflicted during the attack.

As a rule, within twenty-four hours after the attack the patient has entirely recovered, unless he has sustained an injury. Cases occur, however, in which, sometimes for several days after the attack, sensory disturbances, paralysis or weakness, mental confusion, disturbances of speech, etc., are present.

In some cases the convulsive seizure may be very mild, and be followed by drowsiness for a short time, or immediately after the attack the patient may continue his occupation as if nothing had occurred. In some few cases the patient may be able to prevent an attack by taking certain precautions as soon as the aura occurs.

In *petit mal*, or minor epilepsy, the attacks are characterized

principally by a loss of consciousness. The seizures come on suddenly, are rarely preceded by an aura, and usually last only a very short time—from a few seconds to half a minute. The disturbance of consciousness passes off rapidly, and in many cases the patient is unaware that an attack has occurred. The face is usually pale, the body rigid, and the eyes are staring. The patient rarely falls. There are often slight clonic twitchings, especially of the muscles of the face or of the upper extremities. If the patient is engaged in conversation, he suddenly stops talking, and those observing him notice that something peculiar has happened; in a few seconds the patient may continue the conversation, not realizing that it has been interrupted.

If the patient should be engaged in some occupation or be eating at the time the seizure occurs, he may drop his tools, upset objects in his immediate neighborhood, drop the food he is conveying to his mouth, and a few seconds later, when the attack has passed off, he observes with astonishment what has occurred. During the attack the patient may make peculiar noises, blink his eyelids, make peculiar movements with his lips and tongue, etc.

Psychic Epilepsy.—In some cases, instead of a loss of consciousness, a peculiar mental state occurs in which the patient moves about, becomes fretful, is restless, carries on complicated activities, may suddenly run aimlessly about, exposing himself, etc. He has absolutely no recollection of the condition after the attack has passed.

Attacks of minor epilepsy are apt to occur more frequently than major attacks, and in some cases both major and minor attacks occur. Seizures characterized by states of mental confusion, during which complicated activities are carried on, are of infrequent occurrence, and can usually be definitely diagnosed only when they occur in conjunction with either major or minor attacks.

In an individual afflicted with epilepsy there may occur at any time a state in which convulsive seizures may follow one another in rapid succession over a period lasting from several hours to one, two, or three days. This condition is known as *status epilepticus*. This is a very grave condition, and if the

patient survives, it is frequently followed by a period of mental confusion, delirium, weakness or paralysis of a transient nature, or at times, if serious vascular disturbances have occurred during the seizure, the weakness or paralysis may become permanent. In other cases after such attacks sensory disturbances, such as paresthesias, blindness, deafness, etc., occur, which are usually of a transient nature, but may become permanent.

At times, instead of a convulsive seizure, there occurs a disturbed mental state during which the patient may carry on complicated activities, or during which he may become excited, depressed, or delirious; this condition is known as *epileptic equivalent.*

Postepileptic Mental States.—Following a severe attack, or a series of attacks, there may occur any one of the various forms of mental disturbance. These mental disturbances after the epileptic attacks occur very much more frequently than the mental epileptic equivalent. This postepileptic condition may be characterized by confusion, during which the patient speaks disconnectedly, does not seem to have a clear insight into his surroundings, acts peculiarly, cannot control himself, and does not seem to realize the demands made by his surroundings. This state of confusion may last a few minutes, and rarely longer than an hour. It passes off somewhat quickly, and the patient has no recollection of what has occurred during the attack. In other cases after one or several attacks active delirium develops. This delirious condition may last for several hours or days, and during this time the patient may be violently excited and very destructive; he does not seem to feel injuries or pain. These patients frequently act as if they were being driven by some irresistible power; at other times their actions seem to be the result of hallucinations and delusions. Occasionally, instead of maniacal excitement, states of depression or anxiety, with terrifying hallucinations, may occur. In the maniacal stage the patient may commit crimes, and in the depressed stage he may attempt self-destruction.

The symptomatic and Jacksonian epileptic seizures require no special description, being merely evidences of a general disease or of local irritation of the brain.

Between the attacks the mental and physical condition of the patient is often normal. In many cases stigmata of degeneration are present. In some individuals, even between attacks, peculiarities of disposition exist. Epileptics are often of an excitable, irritable, and suspicious temperament. In some there are present a diminution of intelligence and weakness of memory.

The forms of epilepsy that develop early in childhood are often combined with imbecility or idiocy, and those in whom the mind may have been normal at first, may later, as the result of repeated and severe attacks, develop mental weakness.

Course.—The disease usually begins in early life, and the first attack can generally be attributed to some definite cause, but this is not always the case. Between the first and the second attack there usually occurs a somewhat long interval, succeeding attacks appearing at shorter intervals and usually without ascertainable cause. In some cases there is a distinct aura before the attacks, but in many there is no warning, the attacks coming on suddenly, and lasting for a variable length of time. They are followed by mental confusion and sleep, although in some cases the mind of the patient is comparatively clear immediately after the attack. The milder the attack, the more apt is the patient to be mentally clear shortly after.

Attacks occur more frequently during the day, but in some patients the attacks occur only at night, and if they are not very severe, and if the patient sleeps alone, they may remain undiscovered.

Individuals may have both major and minor seizures, or suffer only from the major or the minor attacks. In some patients the seizures occur with considerable frequency for a long time, and then a long interval intervenes during which the patient is free from seizures. Sometimes attacks occur with a certain regularity, or the patient is able to foretell by his feelings, one or two days previously, that he is about to have a seizure.

Acute febrile diseases, injuries, operations, etc., often seem to inhibit the occurrence of the attacks, but shortly after the acute illness is over they reappear. In some few cases an acute illness has seemed to effect a cure.

This disease runs a prolonged course, with more or less frequent repetition of attacks, which occur at indefinite intervals. The attacks may be followed by peculiar mental states and transitory or permanent physical disability, which is often, sooner or later, attended by permanent physical and mental degeneration.

Prognosis.—The prognosis as to complete recovery is, unfortunately, not good. In a few cases in which a definite causative condition can be discovered and removed, the prognosis is good. In those cases in which no cause can be discovered there are between 5 and 10 per cent. of recoveries.

The cases associated with acquired or congenital mental weakness present the most unfavorable prognosis. The longer the disease has lasted, the less chance there is for recovery to take place. The earlier in life the disease manifests itself, the graver is the prognosis. The probability of recovery is not so good in minor epilepsy as it is in the major form. The danger to life in individual attacks is not great, provided the patient is properly protected. Owing to the fact, however, that these seizures may occur at any time and under any conditions, accidental death may occur during an attack. These patients should be advised to be very cautious about exposing themselves to danger.

From 25 to 50 per cent. of patients who develop the status epilepticus die during this state. A large number of individuals who suffer from epilepsy die of intercurrent conditions before they reach the thirty-fifth or the fortieth year.

Diagnosis.—The diagnosis of epilepsy is to be made only after a careful consideration of the history of repeated attacks in which disturbance or loss of consciousness occurs. Epilepsy must be differentiated from simple fainting spells, from convulsive seizures due to a toxic condition, such as occurs in kidney diseases, gastro-intestinal intoxication, etc., from Ménière's disease, and from hysteria. The diagnosis must never be made from the occurrence of a single attack, and it is only the repetition of attacks, with or without apparent cause, that justifies the diagnosis. Repeated convulsive seizures or peculiar mental states in children, when accompanied by evidences of mental degeneracy, usually mean the existence of epilepsy. Attacks

of petit mal, beginning in early childhood, generally present no difficulties in the diagnosis. The differentiation between epilepsy and symptomatic convulsions depends entirely upon the examination, which will reveal the cause of the convulsive seizures. The differentiation between epilepsy and Ménière's disease is made by the constancy of the tinnitus aurium, the disturbance of hearing, and the effect of sudden changes in position in Ménière's disease.

From simple *fainting spells* epilepsy can usually be very readily diagnosed: in fainting spells the face becomes pale, the heart action feeble, and the reflexes are not lost; the patient does not injure himself in falling, there are no convulsions, and the seizure is generally brought on by a sudden mental impression or a sudden change of position: in epilepsy the face is cynanotic or flushed, the pulse full, convulsive seizures or peculiar mental states exist, and the seizure comes on without cause or follows a definite aura; the patient may injure himself or lose control of the bowel and bladder, and the reflexes, particularly the pupillary reflexes, are lost.

The differentiation between *hysteric attacks* and epileptic attacks may in some cases give rise to considerable difficulty. The differentiation is made by a careful inquiry into the facts presented on page 568.

Treatment.—Before undertaking the treatment of a person suffering with epilepsy it is most important to make a careful and thorough examination in order to discover, if possible, the presence of the causes of the attack. Any abnormalities or diseases which are found should be carefully considered and receive proper treatment. The presence of intestinal parasites, of nasal disease, sinus disease, unerupted or carious teeth, phimosis, gastro-intestinal disorders, toxemia, pregnancy, scars, etc., should be carefully examined for, and the patient brought into as good a physical condition as possible. The correction of general diseased conditions or of abnormalities, the elimination of toxic substances, such as alcohol, tobacco, etc., may in some few cases result in bringing about cessation or cure of the attacks; even if this does not take place, it may help in the symptomatic treatment of the disease.

	Epilepsy.	Hysteria.
Cause:	Frequently none.	Generally some emotional condition.
Age:	Usually begins before the twentieth year, and frequently in early childhood.	Usually manifests itself for the first time after the fifteenth year, but may occur earlier.
Sex:	Slightly more frequent in males.	Much more frequent in females.
Conditions under which attacks occur:	Occur under any condition.	Attacks usually occur only when the individual is under observation.
Aura:	Always the same.	Variable.
Abnormal movements:	Tonic, followed by clonic, spasms.	Clonic, followed by tonic, convulsions.
Character of movements:	Purposeless.	Usually result in some definite purposive actions, such as pulling the hair, scratching, biting, etc.
Duration:	Tonic state for a few seconds, clonic state for a few moments.	Tonic and clonic states alternating, lasting from a few moments to several hours.
Appearance:	Cyanotic, followed by a flushed appearance of the face.	Never cyanotic, may be pale or flushed.
Breathing:	No respiration during tonic states, but followed by stertorous breathing, and gradually becoming normal.	No disturbance, at times noisy, shallow, rapid, or slow.
	Bloody froth and saliva dribbling from mouth.	Usually not present.
Injury:	Biting the tongue, cuts, bruises, fractures, etc.	At times scratching of the hands or face or injury to others, rarely to self.
Deep reflexes:	Almost always lost during attack and shortly after.	Generally increased.
Pupillary reflexes:	Reaction practically always lost.	Practically never lost.
Bowel and bladder reflexes:	Frequently incontinence of urine and feces at the onset or during the attack.	Practically no disturbance; the passage of a large quantity of pale urine after an attack.
Following attacks:	Patient recovers rapidly, is confused for a short time, or sleeps for several hours.	Rarely sleeps except as the result of exhaustion after a long and severe attack; usually recovers slowly.
Effects of suggestion:	None.	Frequently reacts to suggestion during the attack.
Temperature:	Increased in severe attacks.	Practically no increase.

The patient should be placed on a light diet; all irritating foods, such as spices, strong coffee, alcoholic liquors, heavy pastries, improperly cooked food, etc., should be avoided, and the diet should consist principally of vegetables, milk, meat in small quantities, fruits, light desserts, the white meat of fowl, fish, etc. It is better to eat frequently, taking small quantities at a time. Salt should be used as sparingly as possible, as it has been found that when salt is restricted less bromid is required to control the attacks.

Exercise may be allowed in moderation, but fatigue, worry, mental overstrain, and excitement are to be carefully avoided. The patient should engage in an occupation that does not expose him to danger; thus he should avoid occupations that require him to ascend ladders or to work on scaffolds, or any employment that requires him to be near moving engines or machinery, or close to water or to fire, etc. He should not follow any vocation that requires constant mental alertness the lapse of which might endanger his own life or the lives of others, such as that of railroad employee, watchman, etc.

Climate.—Extremely hot or cold climates should be avoided, and the patient should guard against unduly exposing himself to extremes of heat and cold.

Hydrotherapy.—Warm baths, followed by cold sponges, keeping the skin in good condition and promoting the general health, do good.

Cases in which the attacks are associated with or followed by marked mental disturbances are better protected in institutions.

Treatment of the Patient During the Epileptic Attack.—In some cases that are preceded by aura the attack may be aborted at times by the inhalation of amyl nitrite, the administration of a dose of aromatic spirits of ammonia, the application of a ligature about the extremity in which the aura is perceived, or by the patient exercising violently. During an attack there is very little that can be done, except to see that the patient is placed in such a position that he cannot injure himself; all tight clothing should be loosened, and, if possible, a cork or some cloth-covered object is to be placed between the teeth, so as to prevent the

patient from biting his tongue. As the attack subsides the patient should be placed in a comfortable position, so that he may go to sleep; no effort should be made to awaken him. If the patient has a succession of attacks, it is advisable to administer purgatives, and if the seizures follow one another, or if there is no return of consciousness, croton oil, one or two drops in a small amount of olive oil, should be placed upon the back of the tongue. Chloral hydrate, in doses of from one-half to a dram and a half, with or without bromid, should be given by rectum. Hyoscin and morphin may be given hypodermically. If these drugs do not control the seizures, blood-letting may be tried or chloroform inhalations given. This last is generally successful in bringing the convulsions to an end, at least temporarily; if, after the prolonged use of chloroform, the convulsions again reappear, a fatal termination may be expected.

Treatment of the Patient Between Attacks.—In most cases, after all that is possible has been done to treat the probable cause and to regulate the patient's life, it is necessary to give medicaments to control the number and severity of the seizures. In those cases in which the attacks occur very infrequently, *e. g.*, one convulsion in one or two years, drugs are not absolutely necessary.

The most useful and effective of all remedies are the bromid preparations. Any one of the bromid salts may be used, potassium bromid seeming to be somewhat superior to the other salts in its effect upon this disease. It is advisable to begin with small doses of the bromid salts, given three times a day, gradually increasing the dose until sufficiently large doses are given to control the convulsions. It is advisable to vary the bromid salts from time to time, and they may with advantage be combined with a bitter tonic, given well diluted, as they seem less likely then to cause gastric disturbances. Such a combination is the following: Potassium bromid, 15 grains; tincture of gentian compound, ½ dram; water, a sufficient quantity, given several times a day.

It is well to try the different salts of the bromids in different individuals, since some bear one salt better than another. The bromid may be given in one dose or in several doses, or one at

night and one in the morning, or two small doses during the day and a large dose at night—one must be guided by the results and the surroundings of the patient as to the best time to give the drug and the size of the dose. If the bromids are not well borne, bromotone, in 5-grain capsules, may be given. The bromids must be given in sufficiently large doses to control the seizures—the smallest doses that will control them should be continued for a prolonged period, and be discontinued only in case of necessity, such as the occurrence of some acute illness, extreme exhaustion associated with mental dulness, physical weakness, gastric disturbances, etc. After the continuous administration of bromids, if no attack has occurred for two years, their administration may be discontinued. In some cases the bromid may entirely suppress the attacks, but in the greater number it merely relieves them, and in some, especially those of the petit mal type, it seems to have no effect. In case the bromids alone do not prove effective, they may be combined with the fluidextract of belladonna, or, in other cases, with tincture of digitalis. In some cases atropin seems to control the attacks. This last should be administered first in small doses, gradually increasing the dose until the physiologic effect is reached. Bromipin, which is potassium bromid combined with oil of sesame, one dram equaling thirty grains of potassium bromid, although possessing a very disagreeable taste, seems to be valuable, owing to its nutritive properties; it rarely causes acne. In some cases chloral hydrate in 5-grain doses may temporarily be added to the bromid preparations, but if its use is long continued it produces cardiac weakness.

Calcium lactate and parathyroid extract in some cases seem to exercise a favorable effect, especially in the attacks of petit mal. Antipyrin, in doses of ten or fifteen grains two or three times a day, also seems to have effect in controlling the convulsions. Borax, ten to twenty grains, taken after meals, is sometimes effective when the bromid preparations fail; this drug cannot be taken in large doses for any length of time as it produces cutaneous eruptions of an eczematous nature.

Surgical Treatment.—The operative treatment of epilepsy is justifiable only when there is distinct evidence of the existence

of some localized cerebral irritative area. This is especially the case if, after injury, there is evidence of a localized cerebral irritation, or if symptoms point to the existence of a localized cerebral lesion that is acting as an irritant, and which is so situated as to be readily accessible to surgical intervention.

Scars or cicatrices upon the skin, or in some other portion of the body, producing epileptic seizures by acting as an irritant, should be carefully sought for and promptly removed. After operative procedures systematic medical and dietetic treatment should be continued for a long time.

Operative procedures are not justifiable in epilepsy unless there are definite evidences of cerebral or reflex irritation.

MYOCLONUS EPILEPSY OF UNVERRICHT

This disease is an exceedingly rare affection, in which epilepsy is present, combined with the occurrence of tonic or clonic spasms in different muscles, which may or may not be painful. The disease is usually seen to occur in several members of the same family, generally beginning in early childhood as a nocturnal epilepsy. The attacks occur at first infrequently, and later at shorter intervals. Sometimes, instead of an epileptic attack, the patient awakens with painful tonic or clonic spasms in the extremities. The disease is chronic in its course, and later on, if the attacks increase in frequency, a stage is reached in which the tonic or clonic spasms of the extremities are more or less persistently present.

Tremor, with fibrillary fascicular contractions of the muscles, at first limited to the extremities, and gradually extending to involve all the voluntary muscles of the body, occurs sooner or later. The tremor varies from time to time, and alternates with lightning-like contractions in the various muscles. The tremor ceases during sleep, and is increased upon exercise or excitement. The knee-jerks are exaggerated. After the disease has existed for some years gradual mental disturbances, characterized by dementia, depression, or excitement, occur.

The prognosis is very unfavorable; after the disease has run a prolonged course the individual dies of general asthenia.

PARAMYOCLONUS MULTIPLEX

This affection is characterized by clonic contractions, affecting mainly the muscles of the trunk and the proximal portions of the extremities. The contractions occur in paroxysms, are usually bilateral, and affect symmetrically situated muscles. As many as from 60 to 100 contractions a minute may occur, the intervals between the paroxysms being of varying duration.

Etiology.—The cause of the disease is in doubt. It occurs more frequently in males than in females, and generally appears in adult life. It may follow fright, anxiety, overexertion, trauma, etc.

Pathology.—Friedreich and others believe this neurosis to be due to an irritation of the nerve-cells of the anterior horn of the spinal cord. Similar symptoms have been observed in auto-intoxications.

Symptoms.—This disease is characterized by the occurrence of bilateral, symmetrically situated contractions of muscles. These spasmodic contractions are limited to the muscles of the trunk, the proximal portions of the extremities, and at times the diaphragm. In no case have the muscles of the forearms and hands or the legs and feet been affected.

The spasmodic paroxysms occur at intervals varying from a few minutes to days or weeks. During a paroxysm the muscular contractions vary in number from 50 to over 100. The paroxysm may last from a half-minute to several minutes. The severity of the muscle contractions varies in different individuals and at different times: in some cases they are so violent that the individual's body is thrown about; in other cases they are so slight that the movements of the limbs are hardly perceptible, only fascicular twitchings of the muscles being evident.

The tendon reflexes are usually increased. There is no disturbance of sensation, and voluntary movements are, as a rule, unaffected. Spasmodic seizures may often be brought about by a tap upon the patellar tendon or by irritation of the skin. Excitement and emotional disturbance increase the tendency to seizures; distraction and occupation decrease this tendency.

In some cases fibrillary tremor of the affected muscles is present during the intervals between attacks.

The **prognosis**, in the majority of cases, is good, as these patients recover rapidly under proper treatment. Relapses are frequent.

Diagnosis.—This disease must be differentiated from chorea, hysteria, epilepsy, tic, etc. From *chorea* the disease is differentiated by the fact that in chorea the spasmodic movements are not symmetrically bilateral, and that they affect the distal portions of the extremities or the face as frequently as they do the proximal portions; moreover, the muscle contractions do not occur in definite paroxysms, but occur irregularly and unexpectedly. Paramyoclonus multiplex is differentiated from *hysteria* by the fact that it is limited to paroxysmal clonic contractions of the muscles of the trunk and proximal portion of the extremities, which can frequently be brought on by tapping the knee-tendon or by inducing irritation of the skin; in hysteria the spasmodic seizures are either more diffuse or more limited in extent, vary from time to time, and are not brought on by tapping of the knee-tendon or irritation of the skin; in addition, there are usually many other symptoms of a sensory or a mental nature.

From *epilepsy* this disease is differentiated by the fact that there is no disturbance of consciousness, and that the spasmodic movements are limited to the proximal portions of the limbs and the trunk.

From *tic* it is differentiated by the fact that the movements in the tics are purposive in character, and very frequently involve the face, and when they involve the extremities, are very apt to affect the distal portions, and that the tics are not brought on by tapping of the knee-tendons or by irritation of the skin.

Treatment.—The administration of sedatives, such as bromid of potassium, chloral, etc., has a quieting effect, and produces a temporary improvement. Tonics, with general up-building of the patient by massage and hydrotherapy, are to be tried. Galvanism applied to the spine seems to be of some benefit.

DISEASES DUE TO PERVERSION OF SECRETIONS OF THE DUCTLESS GLANDS

EXOPHTHALMIC GOITER (BASEDOW'S OR GRAVES' DISEASE)

Etiology.—A neuropathic heredity may be traced in many of the affected individuals. The disease occurs more frequently in women than in men, and develops more often between the twentieth and the fortieth year, but may appear much earlier or later. Neuropathic heredity manifests itself very often as a tendency to vasomotor neurosis in other members of the family, and as nervousness in the individual sometimes long before the onset of the disease.

In a predisposed person exophthalmic goiter may occur without any other direct cause. In many cases the disease is preceded by emotional excitement, worry, injury, physical exhaustion, pregnancy or difficult labor, infectious disease, syphilis, chronic intoxication, etc.

Pathology.—At the present time there is still a difference of opinion as to the primary cause of this disease. Some investigators believe that the primary changes are in the sympathetic or central nervous system, and that a secondary disturbance in the circulation of the thyroid gland occurs, with an increased formation of its secretions, and that, as the result of the action of these substances, many of the secondary symptoms of this disease develop. On the other hand, there are many who believe that the primary disease affects the thyroid gland; that there is an increase in the secretions of this gland, and that, as the result of this toxic substance entering the circulation, many of the nervous and secondary symptoms occur. Careful ex-

amination of cases of exophthalmic goiter shows wide-spread changes in the thyroid and the thymus gland, the cervical sympathetic, the brain, the cranial nerves, and the medulla. In the thyroid gland the changes vary from a simple congestion to a marked hypertrophy of part or of the entire gland.

The colloidal substance is usually diminished in quantity; the vessels are dilated; the connective tissue is generally developed to an abnormal drgree; the lymph-spaces are obliterated, and there is a hyperplasia of the parenchymatous tissue. Very often a hyperplasia or an abnormal persistence of the thymus gland is present. Degenerative lesions have been found in the brain, in the nuclei of the cranial nerves, and in the cervical sympathetic.

Symptoms.—Tachycardia is one of the most constant and one of the earliest symptoms of this disease. It occurs in practically every case, the pulse varying in frequency between 100 and 200 a minute. It is generally small and soft, averaging about 120 a minute. The arteries, especially the carotids, pulsate strongly and visibly; the increased pulse-rate is constantly present. In addition, as the result of the muscular exertion or emotional disturbance, attacks of cardiac palpitation occur in which the rate is increased, and at times arhythmia develops.

Precordial pain is frequently present. On physical examination the heart often shows no abnormalities, although sometimes a systolic murmur is present at the base. The heart is often enlarged, and in advanced stages it becomes dilated, with the development of a mitral insufficiency.

Enlargement of the thyroid gland, which may be limited to one lobe or affect the entire gland, occurs in many cases. The enlarged thyroid (goiter) is soft and pulsating; the superficial veins are usually dilated; a marked thrill can sometimes be perceived by the hand placed above or over the thyroid. A systolic bruit can often be heard an auscultation. The goiter is not painful. The degree of swelling varies from time to time. When increase in the size of the thyroid occurs rapidly, it may produce symptoms of compression and interfere with breathing and with deglutition.

Many cases of exophthalmic goiter occur in which there is little

or no perceptible enlargement of the thyroid. It is important to remember that the thyroid may be diseased even if it is not enlarged.

Exophthalmos, which is usually bilateral, but which may be unilateral, is also a common symptom. Early in the disease it may be limited to one eye, the left one being particularly affected. Throughout the disease it may be more pronounced on one side than on the other. The degree of exophthalmos varies in different cases—sometimes it is very slight, and in

Fig. 158.—Exophthalmic goiter (case of Dr. Randolph Winslow).

other cases it is so marked that the eyelids cannot be completely closed, and the insertions of the eye muscles are visible (Fig. 158).

Tremor is almost always present. It is rapid and vibratory, from 8 to 10 oscillations occurring to the second. It is increased by movement and by emotional disturbances. It is sometimes limited to the extremities, and at times involves the trunk. It does not necessarily persist throughout the whole course of the disease.

Trophic, vasomotor, and secretory disorders also occur in nearly all cases. Discoloration of the skin, urticaria, pigmentation of the margins of the eyelids, falling out and turning gray of the hair, swelling of the eyelids, hemorrhages from the uterus and nose, ecchymosis, etc., sometimes occur early, and generally appear at some time during the course of the disease. Dermographia occurs very frequently. Flushes of heat and cold and slight elevation of temperature are also commonly observed. Hyperidrosis is a very frequent symptom, being absent in only a few cases; the sweating may be partial, and be limited to the hands and feet or head, or it may be general; it varies in degree —in some cases it is but slight, and in others it is so extensive as to necessitate frequent change of clothing. The secretion of urine is often increased; albuminuria and glycosuria occur but rarely.

Digestive disturbances are frequently complained of. Loss of appetite, dryness of the mouth and pharynx, and attacks of vomiting may occur, the last giving rise to much distress. Attacks of diarrhea in which the stools are sudden, frequent, and usually painless, from four to ten or more occurring in a day, and not dependent on the diet, are common. These diarrheas are more frequent upon exertion or after emotional excitement, and generally contribute greatly to the patient's exhaustion.

The general nutrition of the patient is often disturbed. There is a loss of weight, which may be slight, but in some cases is very marked, and occurs in spite of the taking of an increased amount of food.

Physical weakness, varying from a slight degree to one in which the patient is almost unable to move, sometimes occurs. This physical weakness is due partly to the emaciation, but actual motor weakness which affects chiefly the legs also exists. The deep reflexes are usually unaltered, but may be increased or decreased. In some few cases attacks of dyspnea occur, and a dry, spasmodic cough is sometimes complained of. Irregularities and disturbances of menstruation may be present.

The mental condition of an individual suffering with exophthalmic goiter is rarely normal. These individuals are usually restless, irritable, and very excitable. The emotional

tone is most variable: the patients are easily confused, very forgetful, easily embarrassed, etc. In some cases marked psychoses, such as acute delirium, melancholia, excitement, hallucinations, etc., develop, but these are not at all common. Symptoms of exophthalmic goiter may also develop during the course of a psychosis. Sleep is usually disturbed, and insomnia is present in many cases.

Ocular Symptoms.—The ocular movements may be impaired by the exophthalmos, but true paralysis of the eye muscles is very uncommon. A vibratory tremor of the eyeball is sometimes noticed. Ophthalmoscopic examination usually reveals a distinct pulsation of the retinal arteries, but otherwise there are no fundal changes.

Stellwag's sign may be present: The palpebral aperture is abnormally wide, even when there is no exophthalmos, and the patient does not wink as often as the normal individual.

Von Graefe's sign is often seen, and consists of an inability of the eyelid to follow the movements of the eyeball. Thus when the globe is voluntarily moved upward or downward, there is a delay in the movements of the eyelids in the corresponding direction. When looking downward, the superior conjunctiva becomes visible.

Möbius' Sign.—This consists of a difficulty in convergence, and is of value only when there are no errors of refraction. This sign is tested for by having the patient look first at a distance and then at a point close to his nose. When the sign is present, only one eye assumes the position of convergence and the other deviates downward.

Course.—This disease is usually of slow onset, but may develop acutely, especially after violent emotion. In many cases there exists, for some time before the onset of the characteristic symptoms, general weakness, fatigue, and loss of weight. Sometimes before the diagnosis can be made a stage exists in which the individual presents symptoms of a neurasthenic or a hysteric character.

Cases also occur in which some of the leading symptoms are absent; thus forms of exophthalmic goiter occur without enlargement of the thyroid or without the exophthalmos—the

absence of one of these symptoms is not at all uncommon. The disease is usually a protracted one, and may last for many years, with remissions and intermissions. A type also occurs in which the development of the disease is very acute, and which runs an acute course, terminating fatally in a short time, or, after an acute onset, running a chronic course.

Prognosis.—The prognosis depends upon the cardiac condition and upon the state of nutrition. Cases that are of acute development, those which have persisted for a long time, and those that cannot or will not take proper treatment, present an unfavorable outlook as regards recovery. In those cases in which the general nutrition is good and in which the mental symptoms are not very severe; in which the pulse-rate is not excessively fast, and in which the individual is able to receive proper treatment, the prognosis is hopeful, at least for improvement, if not for complete cure.

There is always danger of recurrence taking place when unfavorable conditions are present, such as acute illness, overexertion, undue emotional excitement, pregnancy, etc. Death may occur from uncontrollable vomiting, from profuse diarrhea, from compression of the trachea by the goiter, from acute cardiac dilatation, etc. The rapidity of the heart-beat and the state of the general nutrition are the best guides as to the gravity of the condition. The outlook is worse where the loss of weight is rapid and excessive and where the tachycardia is extreme.

Diagnosis.—In marked cases there can be no difficulty in recognizing the disease from the presence of its cardinal symptoms and signs. The exophthalmos should always lead one to think of retrobulbar tumors, hydrocephalus, and aneurysm. In the absence of exophthalmos and marked enlargement of the thyroid gland this disease may be confused with neurasthenia and hysteria. Between these two diseases and exophthalmic goiter it may be very difficult to arrive at a positive conclusion as to the correct diagnosis. In exophthalmic goiter, in addition to the tachycardia, which is constant, there are usually present some of the accessory symptoms, such as pigmentation of the skin, ocular disturbances, dermographia, vomiting, diarrhea, tremor, etc. In neurasthenia or hysteria the tachycardia is

not so constant; the ocular symptoms occur only rarely; the diarrhea or vomiting, loss of weight, etc., are not so persistent, nor do they have so serious an effect upon the individual's general condition as they do when they occur in exophthalmic goiter. In exophthalmic goiter the mental symptoms are more marked and more constant than they are in hysteric patients. At times it may be difficult to differentiate simple goiter pressing upon the trachea and cervical sympathetic ganglia, giving rise to attacks of dyspnea, hoarseness, etc., occurring in a nervous individual, from a true exophthalmic goiter. In these cases there is usually an absence of tremor, the exophthalmos is apt to be unilateral, and there is an absence of diarrhea, of mental symptoms, of the ocular signs, etc.

Treatment.—It is most important to place the patient at absolute rest. Even in mild or moderately severe cases the patient should be placed in bed, preferably in the open air or in a large, well-ventilated room. All emotional disturbances, worry, and excitement should be guarded against; in order to accomplish this it is generally necessary to remove the patient from his home. Very moderate exercise is sometimes permissible, but fatigue must be avoided. The diet should consist of easily digested and nourishing food. At times it may be advisable to place these patients upon a diet consisting mainly of vegetable foods, to which milk and cream should be added in abundance. Coffee, tea, tobacco, and alcoholic beverages should be forbidden. All exciting diversions are to be avoided, and sexual indulgence is, so far as possible, to be interdicted. Mild hydropathic treatment, such as cool packs, cold rubs, tepid baths, wet packs, etc., may be of benefit in the milder cases. Residence in high altitudes seems also to have a beneficial result.

Electrotherapy.—Stable galvanization of the sympathetic often has a good effect. An electrode (cathode), about one inch in diameter, is placed on the side of the neck at the inner margin of the sternomastoid muscle, below the angle of the lower jaw; the other electrode, which should be larger, is placed on the side of the neck, about the level of the seventh cervical vertebra and a current of two or three milliamperes is applied for several

minutes. This treatment should be persisted in for some time. Care should be taken not to use too strong a current, as this may aggravate the condition. The treatment should be given daily.

Good results have also been obtained by the administration of thyroid extract, parathyroid extract, thyroidin, and the extract of other glands; benefit has also been derived from the administration of a serum prepared by Rogers and Beebe, the antithyroid serum of Moebius.

Certain drugs may be helpful; thus beneficial results have been obtained from the administration of the bromids, iron, and quinin, especially when used in combination with belladonna or ergot. The salicylates, digitalis, strophanthus, atropin, and the iodids must be used with great caution. The bowels should be regulated by mild purgatives. Cold compresses or ice packs applied over the heart may do good. The eyes should be protected and, if irritated, should receive proper treatment. In a few cases improvement has followed the treatment of swellings in the nose and operations upon organs in the pelvis. These later procedures should be undertaken only with great caution and after serious consideration, as they have proved beneficial in only a very small number of cases, and operation may aggravate the condition.

Surgical treatment, in which part of the gland is removed, or in which the thyroid arteries are ligated, is to be recommended in those cases that do not respond to or at least show improvement after six weeks of proper medical treatment.

Before operating upon these patients it is well to consider them carefully as surgical risks: these patients do not do well if they are operated upon late in the disease, after a marked cardiac degeneration and a state of cachexia have developed.

Only partial thyroidectomy should be performed. The proper amount of gland to remove in each case must rest on the judgment of the surgeon. Care should be taken not to remove the parathyroid glands nor to injure the recurrent laryngeal nerve, etc. Surgical intervention is indicated in those cases that are rapidly progressive, in cases with grave nervous and

mental symptoms, and in those in which there is evidence of compression of the trachea, esophagus, etc.

Jonnesco's operation for the removal of the cervical sympathetic ganglia with their cord is believed to exert a favorable influence upon the course of the disease.

In general it may be stated that in all cases except those that are of acute onset, that are rapidly progressive, and that present grave nervous and mental symptoms, an effort should be made, either by adopting a complete or a modified form of the Weir Mitchell treatment, together with electricity, diet, hydrotherapy, etc., favorably to influence the disease. This attempt should be made if no unfavorable symptoms occur for six or eight weeks. If improvement is continuous, the treatment should be persisted in; if, on the other hand, there is little or no improvement, or if there is an increase in the symptoms, surgical measures should be promptly resorted to.

Those cases that are too ill to undergo surgical treatment, and those in which there is a marked persistence of symptoms even after proper operative measures have been instituted, may derive benefit from prolonged exposure of the thyroid gland and the thymus glands to the *x*-rays.

MYXEDEMA—CRETINISM

This condition is characterized by a peculiar swelling of the skin and subcutaneous tissues, secretory and trophic disturbances, mental changes, etc.

Etiology.—The disease may appear in earliest years of life, be of congenital origin, or it may occur later. It affects females more frequently than males. Alcoholism, tuberculosis, syphilis, diabetes, pregnancy, lactation, the menopause, altitude, and certain living conditions seem to predispose to this condition. The disease occurs with frequency in certain localities of Switzerland, Austria, Italy, and France. The direct causes are disease that lead to an inflammatory condition of the thyroid gland, with subsequent sclerosis and atrophy; injury to the gland, with subsequent degenerative changes and insufficient nutrition; tumors of the gland; enlargements of the gland in which there

is a proliferation of the interstitial tissue, with defective function; operations upon the thyroid gland in which the entire gland or too large a part of the gland has been removed, or in cases in which, after operation on the gland, degenerative changes occur. There may be a congenital absence or abnormal smallness of the gland from some unknown cause, etc.

Pathology.—Examination of the skin shows the swelling to be due to a mucous or mucoid infiltration. On examination of the thyroid gland this organ is seen to be usually diminished in size or atrophied. Experimental and clinical cases show that symptoms of myxedema or cretinism are due to a deficiency, a loss of, or congenital absence of the secretions of the thyroid gland. The severity of the symptoms depends upon the extent of the deficiency of the thyroid secretions. The younger the affected individual, as a rule, the more harmful will be the loss of this secretion.

Symptoms.—The onset is usually gradual. The symptoms differ somewhat according to whether the disease occurs in early infancy or somewhat later in life. Deficient or absent secretion of the thyroid gland occasions more severe symptoms in young individuals than in those that are older. As the result of a congenital absence or marked deficiency of the thyroid secretion a condition known as cretinism develops. Physically the stature is dwarfed; the fontanels are large and open; the nose is small and broad; the lips are pendulous; the mouth is open and the tongue is thick and large, being usually too large for the mouth and hence protruding; dentition is delayed; walking and talking are acquired late, if at all; the skin is thick and dry and of a yellowish tint; there is a defective growth of the hair, which is coarse and thick; the mucous membranes are pale and usually swollen; the facial expression is stupid; the eyes are half closed and the eyelids are swollen; the neck is generally short and thick; the abdomen protruding with a low position of umbillicus; the appetite is increased; obstinate constipation; in subclavicular regions and in the axilla fat-like masses are often deposited. *x*-Ray examination shows a defective and delayed ossification and persistence, to an advanced age, of the epiphyseal cartilage; the genital organs

remain infantile, and signs of puberty either do not develop or are materially delayed. Intellectually the condition of these individuals varies from a state of marked idiocy to one in which the lack of mental development is not so marked.

Cases also occur in which the symptoms are not so prominent. In these patients the skin is dry; the hair is sparse; the tongue may be large; the stature is small; the genitalia are poorly developed; there is delay or absence of the signs of puberty; secondary sexual characteristics are absent or poorly developed; the mental development is deficient, the mentality never exceeding that of a normal child.

In the acquired form of myxedema which develops after a pathologic condition that brings about a more or less deficient secretion of the thyroid gland, symptoms of the disease gradually develop. The individual complains of fatigue and weakness in the limbs, a feeling of cold, and more or less diffuse pain. Next a puffiness of the face, hands, and feet develops. The swelling about the chin and the eyelids is due to infiltration of the skin and subcutaneous tissue. The face assumes a heavy expression. The infiltration of the skin appears on the neck and on the extremities; the skin is pale or yellowish, feels cold, is firm and elastic, and does not pit on pressure. The secretion of sweat decreases or ceases, the skin becomes dry and rough, the hair falls out, and fat-like tumor masses may appear in the neck, axillary spaces, etc. The mucous membranes become thickened and infiltrated. There is often a loss of the teeth. The temperature of the body is usually subnormal; headache, forgetfulness, vertigo, weakness, and awkwardness are also present. The voice is usually rough, hoarse, and monotonous. To all these evidences there are added mental changes. These consist most commonly of marked deficiency in the intellect; the memory is impaired; thought processes are slow; irritability or indifference is present, and there is a marked tendency to somnambulism. In some cases sensory illusions are present, and even marked psychoses may occur in this condition.

The characteristic features of myxedema are the swelling, infiltration, dryness of the skin, falling out of the nails, thickening and infiltration of the mucous membrane, hoarse, rough,

monotonous voice, and the mental changes that are usually characterized by simple dementia; a subnormal temperature, an absence, partial absence, or smallness of the thyroid, or an increased size of the thyroid gland. The examination reveals the fact that there is present either a tumor or an increased size of the gland, due to a proliferation of interstitial tissue. Albuminuria and glycosuria occasionally occur.

Course.—This disease usually develops slowly and runs a progressive course. In cretinism the first evidences are apparent after weaning or within the first two years of life. Unless treated, the symptoms become frankly evident within the first few years. In young individuals or adults the symptoms may begin shortly after an operation on the thyroid or following an infectious disease, injury, exposure, labor, etc.; or they may not manifest themselves for several months after these exciting causes have taken place. General physical weakness, intellectual torpidity, changes in the skin, and subnormal temperature develop. The face loses expression, and usually the entire body becomes larger, due to the deposit of fat. Remissions may occur.

Prognosis.—In cretinism there is, as a rule, no tendency to spontaneous improvement. Many of these cases die in childhood. Under proper treatment, however, marked improvement takes place. The intellectual progress is much slower than are the physical changes. The earlier the treatment is begun, the more hopeful is the outlook.

In cases occurring in young adults the prognosis is grave, death being generally due to some intercurrent disease, of which pulmonary tuberculosis is the most frequent. Unless properly treated, death occurs either from an intercurrent disease or from pronounced cachexia.

The prognosis depends upon the integrity of the parathyroid glandules, upon the amount of the thyroid gland removed, and upon the age of the individual. If symptoms of tetany develop along with the symptoms of myxedema, the outlook is very grave. In those cases in which too much of the gland has been removed, or in which, after operation, sclerotic changes take place in the gland, in young adults the outlook is not favor-

able; in older individuals, with proper treatment, it is more hopeful.

Diagnosis.—The condition is diagnosd without difficulty. The combination of the characteristic symptoms, the changes in the skin, the facial expression, the increased weight, the general weakness, the mental feebleness, etc., make the condition one easily recognized. It is always important to differentiate the infiltration of the skin from an edematous state of the skin, as the latter may occur in cardiac and renal diseases, which may also be accompanied by mental changes. A careful examination, together with the history of the case, will usually enable one to differentiate a true cardiac or renal disease from myxedema.

Sporadic and endemic cretinism must be differentiated from mongolian idiocy, achondroplasia, congenital adiposity, and a combination of idiocy with infantile dwarfism.

As a rule, the typical skin changes are absent in these diseases. The *x*-ray examination of the bones shows the lack of ossification, with persistence of the epiphyseal cartilage in cretinism and the absence of these findings in the other conditions. The typical facial expression in cretinism, the thick lips, the protruding tongue, the short neck, etc., are in marked contrast to the characteristic appearance in these other diseases.

Treatment of Cretinism.—In the endemic form the prophylactic treatment is important. In localities where this disease exists improvement of hygienic surroundings and pure drinking-water are important. Owing to the fact that heredity certainly plays some part in the transmission of this disease, it is advisable that those families in which the disease occurs should intermarry only with families that are free from the taint. Upon the appearance of the first symptoms the patients should be sent away from the locality in which the disease exists into one where it does not occur. Treatment should be undertaken as early as possible—the extent of the improvement is dependent chiefly upon the age at which treatment is instituted. Improvement is possible even when growth has ceased.

The patient should be placed amid as favorable hygienic surroundings as possible, and since these patients usually suffer

from cold, they generally do better in a moderately warm climate. The diet should be regulated and constipation rectified.

Thyroid extract, given in the form either of freshly prepared liquor thyroidei or of dried thyroid glands, is the chief remedy to be employed. The thyroid extract should be given at first in small doses, beginning with one grain of the dried gland or five minims of the liquor, given at first once a day, and then two or three times a day, and then increasing the dose until the effects begin to appear. Usually within a few days after the administration of the proper doses the skin assumes a more natural condition, there is a return of the secretion, cyanosis disappears, and there is a gradual loss of weight. Later there is rapid growth of the extremities; the tongue becomes smaller, the muscles grow stronger, the amount of urine increases, the individual manifests more interest in his surroundings, and there is a gradual disappearance of the apparent dementia. The improvement is most marked in younger patients; in older patients the treatment has little effect upon the mental condition. If the dose of thyroid extract is too large, dangerous symptoms may occur, of which the most common are tachycardia, diarrheal attacks, convulsions, general weakness, etc.; consequently great care should be taken in the administration of this drug, and upon the first appearance of symptoms pointing to overdosage its administration should be discontinued for several days, and the treatment then resumed with smaller doses.

The Treatment of Myxedema.—The treatment of this disease is very simple and certain, and the effect is most marked. The extract of the thyroid gland of sheep, in proper doses and gradually increased, should be given, care being taken to avoid inactive or decomposed preparations.

Small doses are given at first and gradually increased. Three or four days after administration the effect of the thyroid extract manifests itself. The body weight diminishes, the peculiar thickening of the skin disappears and it becomes more moist, the pulse is somewhat more rapid, the temperature becomes normal, and there is a marked improvement in the mental symptoms. It is well to begin with two grains two or

three times a day, gradually increasing to three, four, or five grains given three, four, or five times a day. Upon the appearance of symptoms of hyperthyroidism the thyroid extract should be discontinued and its use not resumed for several days, and then the treatment taken up again, beginning with small doses.

The diet should be varied, but should consist chiefly of vegetables. Tonics may be given. The general hygienic conditions of the patient should be properly supervised. In both cretinism and myxedema it is necessary to continue the administration of thyroid gland indefinitely; generally it is wise at intervals to discontinue the administration of the gland for a short time and then to return to it again.

TETANY

This disease is characterized by bilateral intermittent tonic spasms, which are usually painful. They affect principally the hands, and sometimes both the upper and the lower extremities. They are, as a rule, unaccompanied by any disturbance of consciousness.

Etiology.—This disease occurs more frequently in children than in adults, and very rarely in old age. It affects males more frequently than females. Certain occupations seem to predispose to this condition; thus shoemakers and tailors are prone to be affected, probably owing to a poison taken into the system from some article used in these trades. It sometimes occurs in women during pregnancy, lactation, or the puerperal state. It occurs more frequently during the early spring months in children who have been living amid bad hygienic conditions, who are suffering from rickets, gastro-intestinal disease, intestinal parasites, chronic pulmonary disease, and in those who are poorly nourished. It may also occur after typhoid fever or following any of the infectious diseases, after removal of the thyroid gland with injury to the parathyroids, dilatation of the stomach, after chronic intoxication from alcohol, lead, chloroform, or in nephritis, etc.

Pathology.—At the present time tetany is almost universally attributed to an absence or an insufficiency of the secretions of

the parathyroid glands. It is believed that the rôle played by toxemias, whether of internal or of external origin, by reflex irritation, etc., becomes an active one in the presence of an insufficient secretion of the parathyroid glands. No constant pathologic changes are found; in some cases hyperemia and circumscribed hemorrhages, cloudy swelling of the ganglion-cells, etc., were found in the spinal cord; these were absent in others. In still other cases changes were found in the cortex of the brain and in the thyroid glands.

Symptoms.—As a general rule, before the occurrence of muscular spasms there are pains in the limbs, sometimes headache, a general feeling of illness, and very often paresthesias. Either with or without these symptoms a cramp-like spasm occurs in which, generally, the muscles of the hands and fingers are chiefly, and sometimes solely, affected. The attitude of the fingers and the hands in these cases is characteristic: the middle and terminal phalanges are extended, and the basal phalanges are strongly flexed, the fingers being pressed together, and the thumb turned in and covered with the other fingers.

In the more marked cases the hand is flexed at the wrist and the forearm upon the arm; the arm is usually held close to the thorax. When the fingers are pressed closely together over the thumb the hands assume a cone shape which resembles the writer's or accoucheur's position. Sometimes the fingers, instead of being flexed, are spread out, and at times the hand assumes a claw-like position. The lower extremities are also often involved, and when they are, the flexors of the toes become tonically contracted, the toes being strongly flexed, the arch of the foot accentuated, and the foot extended at the ankle-joint, giving rise to the equinovarus position. All the muscles of the leg may be affected; in this case the legs are stiff and may be either abducted or adducted. The muscles of the trunk, tongue, pharynx, and larynx may be involved, and deglutition, respiration, etc., may be affected; this occurs, however, only in very severe cases.

These spasms usually come on suddenly and subside gradually, but recur in a series of attacks. The spasm is generally attended by pain in the affected muscles. In most cases the spasms are

limited to the muscles of the fingers and hands; in other cases they are very much more extensive; they also vary in intensity, in some cases lasting only a few minutes, being very mild and easily overcome, whereas in other cases they are much more severe and may last from a few minutes to several hours or even days.

After the spasm has subsided the paresthesia and sense of muscle tension may remain for some time. In some cases after the attack has passed off muscle weakness remains. During the attack the mind is not affected, but in cases in which a marked toxemia is present there may also be mental confusion. In children it is not uncommon for tetany to be associated with convulsive seizures. The attack is often attended by a slight rise of temperature—100° to 102° F.—and an increase in the pulse-rate.

The spasms may be separated by intervals of varying duration, but, as a rule, they follow each other in quick succession, and in some cases the muscles are never completely relaxed.

In addition to the spasms, which are the most important symptoms of the disease, a number of characteristic signs occur:

Trousseau's Sign.—Trousseau found that pressure on a nerve or a nerve plexus in the interval between spasms will bring on a spasm of the muscles supplied by the compressed nerve. The spasm may not follow for from a half minute to several minutes after the pressure is exerted. Trousseau's sign is not found in any other disease, and is pathognomonic of tetany.

Chvostek's Sign.—This sign depends upon an increased mechanical excitability of the motor nerves. Slight percussion upon a motor or mixed nerve may produce contraction in the muscles which they innervate; this is most readily brought out by slight percussion over the seventh nerve, when there occurs the so-called face phenomenon, which manifests itself by marked twitchings or spasmodic contractions of the face. This sign is not constant, and occurs in diseases other than tetany.

Hoffman's Sign.—This sign depends upon an increased mechanical and electric excitability of the sensory nerves. In

an affected individual slight pressure on a sensory nerve produces paresthesias in the region supplied by the nerve, whereas similar pressure in a normal individual would produce only a local sensation. The sensation caused by pressure on these nerves in an individual with tetany is similar to that which occurs in a normal individual who srikes the ulnar nerve, causing numbness, paresthesia, etc.

Stimulation of the sensory nerves by the galvanic current, if of sufficient strength, produces sensation in the nerve and radiates into the surrounding structures: in tetany these sensations and the radiating sensation occur with very much weaker currents. This symptom is also present at times in neuropathic individuals.

Erb's Sign.—Increased electric excitability of the motor nerves. This sign is present in almost every case. Mild galvanic stimulation of a motor nerve gives rise to prompt and marked muscle contraction; this contraction is usually prolonged. Slightly stronger currents give rise to a tetanic contraction which may not subside until the current is interrupted. Increased excitability to the galvanic is more commonly present than to the faradic current. The ulnar nerve is the nerve which most frequently gives a positive response to this sign, although other nerves will also respond positively.

Among the unusual symptoms that occur in this disease are hyperidrosis, especially after attacks; albuminuria, glycosuria, redness and edema of the skin, urticaria, herpes, falling out of the nails, loss of the hair, and in some few cases paralysis, especially of the muscles of the sacral, lumbar, and pelvic regions.

The reflexes are usually normal, but may be increased or decreased. No objective sensory disturbances are present. In tetany following removal of the thyroid glands, epileptic conditions sometimes occur. Occasionally tetany may be combined with myxedema, exophthalmic goiter, etc.

Course.—The entire disease may be marked by but a few attacks, or may last for several weeks or months. Usually before the attacks come on there is a peculiar sensitiveness of the nerves and paresthesias occur; these are followed by the spas-

modic attacks, that may last for from a few moments to several days. The attacks may occur every day or several times a day, or may be more or less continuous, or they may recur only after more or less prolonged intervals. The individual spasms may last for from a few minutes to several hours. The disease may run its course in a few days, during which one or more attacks may occur, or it may persist for weeks or months. Chronic forms occur in which remissions are present and which may last for many years.

Prognosis.—In the majority of cases the prognosis is good. In those cases in which tetany occurs after removal of the thyroid gland, or when it occurs in persons suffering from gastric dilatation, the outlook is not so favorable, although even under these conditions recovery is possible. When tetany occurs in children who are suffering from gastro-intestinal diseases, death frequently takes place from the primary disease. When the respiratory muscles are affected, tetany may prove fatal, particularly if the diaphragm is involved. Frankl-Hochwart states that tetany occurring in pregnant women presents a grave prognosis.

Diagnosis.—This condition is usually recognized without difficulty, as the spasms are characteristic. At times the condition must be differentiated from *tetanus:* in the latter disease the spasms usually affect first the muscles of the jaw and neck, are generally intermittent, and, as a rule, there is an elevation of temperature. From *hysteria* tetany is differentiated by the absence of the Trousseau and Erb signs, and by the fact that in hysteria the spasmodic seizure is often unilateral.

Treatment.—It is important to make a careful examination to discover the possible cause of the condition. Free movement of the bowels is to be brought about by the administration of castor oil, calomel, and rarely by salines. The nutrition of the patient should be carefully looked after, and the diet regulated. Water should be taken freely, and if there is evidence of gastro-intestinal putrefaction, this should be corrected by castor oil, modification of the diet, and gastro-intestinal antiseptics, such as zinc sulphocarbolate, salol, resorcin, etc. In those cases occurring during pregnancy it may be necessary

to interrupt the gestation. In those occurring during lactation it is advisable to wean the child. If the condition occurs after operations upon the thyroid gland, the administration of thyroid and parathyroid extract is indicated. Many cases do well on calcium lactate, the doses of which is to be regulated according to the age of the patient.

In all cases it is important carefully to treat the patient's general condition, and to make every effort to remove or to relieve the underlying cause.

MYASTHENIA GRAVIS

Etiology.—Females are more frequently affected than males. The disease usually appears between the twentieth and the thirtieth year, but may occur earlier or later. Overexertion, emotional excitement, the infections, especially grip, along with Graves' disease, pregnancy, etc., are among the common coexisting or exciting causes of the disease. In many cases an increased size of the thymus gland has been noticed, and in some cases tumors of this gland have been found. The condition is believed to be due to some error of secretion of the ductless glands, or to a toxic agent of unknown origin that has a selective action upon the muscles. Examination of the muscles reveals numerous collections of lympathic cells more or less constantly present along the course of the lymphatics and about the blood-vessels.

Symptoms.—The disease is characterized by abnormal fatigue of the muscles after exertion. It usually manifests itself in ptosis or a form of strabismus, producing double vision, followed by weakness of the muscles of the face, tongue, pharynx, larynx, etc. This weakness evidences itself in difficulty of speech and in chewing, in swallowing, and in using the lips. The weakness usually extends downward to the arms, hands, and legs. This weakness is at first characterized by the fact that after rest it disappears, and the patient is apparently normal, only to recur on using the part; so that at first the patient may be able to talk plainly, chew well, or see clearly; after using the eyes ptosis and strabismus develop, and he sees double; after chewing

for a short time he tires and uses his hands to aid his jaws in their movements. Later on the disease becomes so marked that a few movements completely exhaust the patient. As the disease progresses a paralysis of the muscles of the eyeballs, face, and mouth develops, lending to the face a mask-like appearance that closely resembles that seen in bulbar palsy. There is usually some dribbling of saliva from the mouth, the speech becomes low and very indistinct, and shortness of breath develops. Failure of respiration and a terminal pneumonia are generally the cause of death. Increased pulse-rate and occasional rise of temperature frequently occur. No sensory disturbances are present. The affected muscles do not show fibrillary tremors nor undergo atrophy. Muscle tone is usually about normal; the tendon and skin reflexes are normal or increased. Examination of the muscles with the electric current at first shows nothing abnormal, but if exercised continuously by either the galvanic or the faradic current, they show a progressively decreasing contractility, finally failing to react to either current. This is called the myasthenic reaction, and it is characteristic of the disease.

Along with the disease there is, as a rule, some depression, or the patient may be very emotional and hysteric. Although the disease usually first attacks the muscles of the face or of the upper extremities, it may begin in the legs and extend upward. The distribution of the symptoms is generally bilateral, although commonly one side is affected more than the other. The onset of the disease is somewhat variable: in some cases the condition develops within a few days or weeks, and in others its progress is much slower.

Course and Prognosis.—This disease runs an irregular course. In some cases a fatal termination occurs within a very short time; in others it takes place within one or two years; in still others there are marked remissions and intermissions, the disease being influenced by treatment, and death not taking place for many years. In a few cases recovery apparently takes place. The prognosis seems to be better in those cases that begin in the extremities.

Diagnosis.—The diagnosis is made from rapid fatigue of the

muscles on exertion; from the appearance of the myasthenic reaction; from the development of double vision, ptosis, difficulty in speech and in swallowing, rapid fatigation of the facial muscles and of the muscles of the limbs, head, and neck, and especially of the muscles of the jaw. The absence of atrophy and of fibrillary tremors, as well as of sensory disturbances, is also a guide to the diagnosis. The condition is to be differentiated from hysteria and bulbar paralysis. From *hysteria* it is differentiated by the fact that strabismus is never present unless as a congenital defect, and then it does not disappear after rest. The difficulty in swallowing in hysterics is caused by spasms of the muscles of the throat, and food is actively rejected through the mouth, whereas in myasthenia the paralysis of the throat leads to regurgitation of food through the nose; if food is swallowed, it may enter the larynx, giving rise to serious respiratory disturbances. In hysteria the patient speaks in a low tone or in a whisper, the articulation being good, whereas in myasthenia the speech is slurring, indistinct, and due to weakness of the muscles of the tongue, larynx, and face. Dyspnea, if present in hysteria, is not attended by cyanosis or physical signs of pulmonary congestion; in myasthenia there are usually cyanosis and accumulation of fluid in the throat and lungs. The myasthenic reaction is never present in hysteria, but is rarely absent in myasthenia.

From *bulbar paralysis* this condition is differentiated by the fact that in bulbar paralysis the eye muscles are usually the last to be involved, whereas in myasthenia they are among the first. In bulbar paralysis atrophy always occurs after the disease has existed for a short time, but practically never occurs in myasthenia. In bulbar paralysis we have the reaction of degeneration: in myasthenia, the myasthenic reaction; in bulbar paralysis there are usually fibrillary tremors, which are absent in myasthenia; in myasthenia we have an early involvement of the extremities, whereas in bulbar palsy this occurs only when the bulbar symptoms are the early manifestations of an amyotrophic lateral sclerosis, and in this condition there are the spastic lower extremities, with a marked increase in the tendon-reflexes. In bulbar palsy the symptoms are not improved by

rest, whereas the contrary is the case early in myasthenia. The symptoms of bulbar palsy usually come on slowly; in myasthenia gravis they are of much more rapid development.

Treatment.—The most important part of the treatment is to shield the affected muscles from all fatigue. The patient should be placed at rest in bed in a quiet room, and not be allowed to talk or to use the eyes. Nutrition should be maintained, and the diet should be as diversified as possible, but so prepared as to require the least possible exertion in mastication and swallowing. The bowels should be kept open. The patient's mind should be diverted, and suitable tonics administered. The effect of repeated prolonged exposure of the thymus and thyroid glands to the *x*-ray may be tried.

DISORDERS OF THE PITUITARY BODY

The pituitary body or gland consists of two portions—the pars anterior, which is developed from the pouch of Rathke, which originates from the buccal cavity, and a pars posterior, which develops from the anterior cerebral vesicle, and is connected with the third ventricle by the infundibulum. Between the pars anterior and surrounding the posterior lobe there is an epithelial tissue which is of different histologic appearance from the lobe. This tissue is called the pars intermedia. We therefore have the hypophysis cerebri, consisting of three structures, the anterior lobe, and the pars intermedia, which are of epithelial origin, and the posterior lobe, which is of neural origin.

The posterior lobe is poorly supplied with blood-vessels, whereas the anterior lobe is highly vascular.

The anterior lobe is made up of cells that have a peculiar staining reaction, the difference being thought to represent different stages of activity in the same type of cells. The active cells seem to distribute themselves in the neighborhood of the thin walls of the venous sinuses into which they doubtless discharge their secretions. In the pars intermedia cells are found that have a tendency to secrete colloid, resembling in appearance the secretions characteristic of the thyroid gland.

Under certain circumstances these cells seem to invade the

posterior lobe, into which the product of their secretion is discharged, and through this structure toward the infundibular cavity, to find its way finally into the third ventricle.

The posterior lobe itself contains no true nerve-cells, being made up of the ependyma and neuroglia tissue. Whether the posterior lobe actually has a secretion of its own, or whether it serves to modify or merely to transmit the secretion from the pars intermedia, are questions that still await solution.

Functions of the Hypophysis Cerebri.—The anterior lobe secretes a substance that apparently enters the blood and influences skeletal growth. An increase of the secretion of the anterior lobe, if it occurs during infancy, brings about a condition known as gigantism; when it occurs later in life, a condition known as acromegaly results.

The secretion of the posterior lobe seems to be more closely allied to the processes of tissue metabolism and to the activity of the renal and vascular system. In diseased conditions of the pituitary gland there may be combinations of inactivity of the posterior lobe, with overactivity of the anterior lobe; of overactivity of the posterior lobe, with inactivity of the anterior lobe; of overactivity of both lobes, or of deficient activity of both lobes—these combinations giving rise to a variety of symptoms. Further modification of the symptoms is dependent upon whether these various disturbances of the functions of the gland occur before or after adolescence.

Etiology.—Inheritance may have some effect in predisposing to diseased conditions of the pituitary gland. Defects are responsible in a certain number of cases, and infectious diseases and injury are also causative factors. Adolescence, puberty, and pregnancy are the periods of life at which disturbance of this gland is more apt to occur. Abnormal activity or diseased conditions of other ductless glands may bring about abnormal activity of the pituitary gland.

Symptoms.—The symptoms of disorders of the pituitary gland may be divided into various groups: First, symptoms that are due to disturbance of the secretions of the gland itself; secondly, those symptoms that are due to an abnormal physical state of the gland bring about local or general cerebral symptoms,

and, thirdly, symptoms due to a disturbed activity of the other ductless glands.

Symptoms Due to Disturbance of the Secretion of the Pituitary Body.—The glandular manifestations occurring before adolescence give rise to the following types: There may occur gigantism, with abnormal and premature sexual developement; or gigantism with an infantile condition of the sexual organs. Another type occurs which is described by Fröhlich, in which there is a stunting of the growth and an infantile development of the genitalia. Other types occur in which adiposity is a common clinical symptom, and which are accompanied by delayed or deficient genital development, and in which there is either a decrease or an increase in the skeletal development.

When these disturbances occur after adolescence, an increased secretion of the anterior lobe gives rise to acromegaly, which may or may not be accompanied by symptoms of an increased or decreased secretion of the posterior lobe, this giving rise, in case of increased secretion of the posterior lobe, to a decreased carbohydrate tolerance, emaciation, high blood-pressure, polyuria, irritability, etc. In cases of decreased secretion of the posterior lobe there occur adiposity, increased carbohydrate tolerance, mental heaviness, subnormal temperature, and regression of the sexual characteristics.

In cases in which the disturbance of secretion is principally in the posterior lobe, there occur, when there is an increased secretion, irritability, restlessness, increased blood-pressure, and decreased carbohydrate tolerance; if this occurs before adolescence, a premature and abnormal sexual development takes place.

If there is a decreased secretion of the posterior lobe, an adiposity occurs that is generalized, involving the internal organs as well as the subcutaneous tissue; the deposit of fat is especially marked in the anterior abdominal wall and in the liver, where the cells are replaced by globules of fat. Increased carbohydrate tolerance takes place, the temperature is subnormal, the pulse is slow, there is mental heaviness, and if this condition develops before adolescence, there is a delayed or deficient development of the sexual organs, together with

absence of the pubic and axillary hair. If the decreased secretion takes place after adolescence, there is, in addition to the other signs, a regression of the sexual characteristics.

Other cases are seen that may be attributed to disturbed secretion of this gland, in which other combinations of symptoms occur.

Cutaneous and subcutaneous changes occur in increased and decreased secretion of the gland: In increased secretion there are external thickening of the epidermis, increase in the size of the hair-follicles, and hypertrophy of the papilla, together with enlargement of the secretory glands. In decreased secretion the skin is smooth and transparent and free from moisture, with an absence of the pubic and axillary hair.

Carbohydrate tolerance is a most important symptom, especially from a diagnostic standpoint: an increased carbohydrate tolerance is found to be associated with a deficiency of the posterior lobe secretion; a decreased carbohydrate tolerance and glycosuria are usually found where there is an increase in the secretion of the posterior lobe. The polyuria associated with disorders is probably due to an increased secretion of the posterior lobe. Polyuria and glycosuria occurring after cranial injury, according to Cushing, are due largely to disturbed activity or injury of the pituitary body. Variations of the temperature occur in many cases. A subnormal temperature generally occurs together with other symptoms that are characteristic of hypopituitarism. Blood-pressure changes are also common, a low blood-pressure and slow pulse occurring in hypopituitarism.

Psychic disturbances are present in many patients; thus with an increased secretion there may occur restlessness, wakefulness, inattention, uncertainty, irritability, and varying grades of intellectual development; with hypopituitarism there occur more or less drowsiness, intellectual slowness, and at times irritability and varying degrees of intellectual development; a psychosis varying in severity may occur in either condition.

Symptoms due to gross physical alteration of the pituitary gland may also occur. These symptoms are divided into two

groups: The first group is caused by a disturbance of intracranial pressure, giving rise to symptoms of general increased cranial pressure; of these, headache is the most common and important symptom; choked disc is present in a number of cases. Venous stasis may also be present, as manifested by fulness and tortuosity of the small and large veins of the forehead and scalp, vomiting, mental slowness, drowsiness, convulsive seizures, etc.

A second group of symptoms may also occur, due to direct irritation and compression or destruction of the structures in the immediate vicinity of the pituitary body. Bitemporal headache, which is severe and persistent, but which may cease after a time, is a very characteristic symptom. Photophobia associated with deep orbital discomfort and sensitiveness of the eyes on pressure is another frequent complaint.

x-Ray examination of the sella turcica may reveal deformities or marked alterations. Atrophy of the optic nerve may occur early, and disturbance of vision and distortion of the visual field are common. Neighborhood symptoms can be demonstrated very early in most cases in which enlargement of the pituitary body occurs.

Abnormal pupillary conditions, oculomotor palsy, nystagmus, and symptoms pointing to irritation or destruction of the temporal lobes are also frequently observed. Mental symptoms pointing to irritation or destruction of the frontal lobes are present in some cases. A fact to be emphasized is that epileptic seizures may be among the early and constant symptoms of this disease.

Course.—The course of the condition depends a great deal upon the cause of the pituitary disease. In those cases in which there is a perversion of the glandular secretion dating back to early life, the course is generally chronic and slowly progressive; in those cases due to tumor the course is usually slowly progressive, depending upon the rapidity of growth of the tumor. In cases due to injury or in those following infectious diseases with secondary involvement of the gland the onset is either acute or subacute, the severity of the symptoms depending upon the extent of involvement of the gland. In this latter group of

cases the condition is usually progressive for a time, after which the symptoms may either disappear entirely or remain more or less permanently present.

Prognosis.—The prognosis depends to a great extent upon the cause. In those cases in which the disease is due to a perversion of the secretions, either in early life or in adult life, the outlook as to recovery is unfavorable. In these cases the disease lasts many years, the patient dying from an intercurrent disease or of a progressive weakness and cachexia. In those cases in which the disease is due to a tumor the outlook is also unfavorable, owing to the difficulties surrounding operative interference, and to the impossibility of completely removing the growth without also destroying the gland.

In cases due to injury or inflammatory conditions of the gland the outlook is somewhat more favorable; in many cases there is a spontaneous disappearance of the symptoms, and in others there is only a partial interference with the activities of the gland, and the disease is thus present only in a milder form.

Diagnosis.—A diagnosis of a diseased state of the pituitary gland can be made only after a very careful examination of the patient, which should include an *x*-ray examination of the sella turcica, of the bones of the face and head, and of the bones of the extremities. The degree of carbohydrate tolerance, the results of examination of the eye-grounds and field of vision, together with the history of the changes in the skeletal growth or of developmental disturbances, premature or delayed development of the genital organs and functions, etc.

Conditions arising from disturbed secretion of the pituitary must be differentiated from myxedema, elephantiasis, hypertrophic pulmonary osteo-arthropathy, osteitis deformans, tumors of the brain situated in other regions, and infantilism due to other causes.

Treatment.—The treatment of diseased conditions of the pituitary gland is dependent upon the cause. In those cases in which the disease is due to the presence of a tumor, treatment is chiefly surgical—to relieve general pressure disturbances subtemporal decompression is indicated; to relieve the symptoms due to irritation or compression of the neighboring struc-

tures, operative measures for the removal of the growth are indicated.

In states of hypersecretion partial removal of the gland may be of benefit, and in cases of hyposecretion transplantation of the pituitary glandular tissue should be attempted.

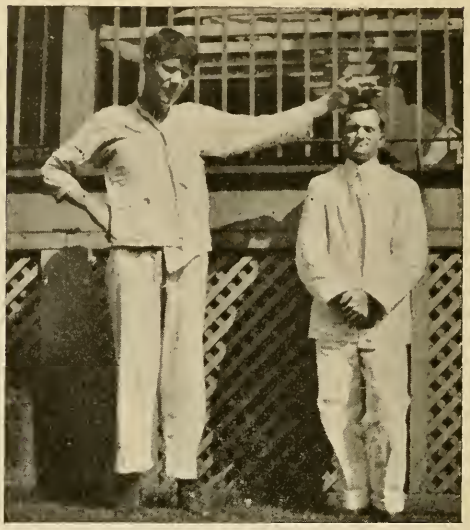
Medical Treatment.—In those cases in which there is evidence of a deficient glandular secretion extract of the pituitary gland may be tried. The administration of the pituitary extract, even when given in large doses, in these cases does not always improve the patient's condition. The extract of the gland may be given by mouth or by hypodermic injection. The administration of the extract hypodermically, either subcutaneously or, better, intravenously, is more apt to be followed by beneficial results than when given by the mouth. Glandular transplantation may be attempted. Prolonged exposure to the *x*-rays has also been recommended as controlling the advance of pituitary growths, and should be used along with surgical measures.

GIGANTISM

This condition is characterized by abnormal growth of the body, which is usually accompanied by physical and mental abnormalities, and is due to a hypersecretion of the anterior lobe of the pituitary body occurring before puberty.

Etiology.—In some cases the gigantism follows infectious diseases, intoxications, trauma, tumors of the brain, etc. Many cases occur for which no apparent cause can be found. The disease occurs more frequently in males than in females.

Symptoms.—The most striking symptom of this disease is the excessive size of the body (Fig. 159). This increase in the size of the body always begins before adolescence, and sometimes in very early childhood. These individuals may reach a height of nine or ten feet, but are usually not taller than seven feet. Along with the abnormality of size a number of different symptoms may occur. In some cases muscular weakness exists; in some there is an infantile development of the genitalia; in others there is abnormal development of the genitalia, with premature sexual manifestations. The mentality of these



A B

C

Fig. 159.—*A*, Gigantism and acromegaly; *B*, normal; *C*, note shape of hands in patient *A*.

patients may be below normal. In addition to these symptoms there may be headache, visual disturbances, epilepsy, polyuria,

glycosuria, increased carbohydrate tolerance, etc. In gigantism after the period of adolescence there may occur changes that are characteristic of acromegaly; usually these changes take place in successive periods, but occasionally the pathologic changes that give rise to these two conditions may take place simultaneously.

Course.—This disease usually runs a chronic course. In some few cases the onset is acute, and the course may be somewhat rapid.

Prognosis.—The prognosis is grave. The condition is usually progressive, the individual generally dying from some intercurrent disease, particularly pulmonary tuberculosis. In a few cases the condition may become spontaneously arrested.

Diagnosis.—The diagnosis is made from the abnormal size of the individual, the delayed or abnormal genital development, the presence usually of mental retardation and of headache, visual disturbances, etc.

Treatment.—Treatment is very unsatisfactory. In cases in which a tumor or an enlarged pituitary body is present surgical measures, such as subcellular or subtemporal decompression, partial removal of the gland, etc., may be tried, as may also prolonged exposure of the pituitary gland to the *x*-rays.

ACROMEGALY

This disease, which has been expecially described by Marie in 1886, is characterized by an enlargement which involves both the soft and the bony structures of the distal ends of the limbs, face, etc.

Etiology.—Heredity seems to play a part, but certainly not an important one, in the causation of this disease. Injury, infectious diseases, emotional disturbances, chronic intoxications, etc., have all been assigned as etiologic factors. Males and females are affected about equally. The condition occurs most commonly in young adults and in middle life. It is conceded now almost universally that this disease is due to a diseased condition of the pituitary gland.

Pathology.—The affected bones show a thickening of the periosteum, new formation of periosteal bone, and thicken-

ing of the tendinous and aponeurotic insertions. Conditions resembling arthritis deformans are present in the joints. The skin covering the affected parts is thickened and hypertrophied. The subcutaneous tissues, glands, vessels, etc., also show a

Fig. 160.—Acromegaly—*x*-ray of skull of patient shown in Fig. 161. Notice enlarged sella turcica, prominent frontal ridge, and projecting lower jaw.

hypertrophy and the thymus is sometimes enlarged. The thyroid gland is usually small, atrophied, or shows goiterous degeneration.

The most persistent change found is the involvement of the

pituitary gland. This is usually hypertrophied and swollen, or a tumor of the gland may be present. Hypertrophy or tumor of the gland may cause irritation or compression of the adjacent parts of the brain, erosion, and enlargement of the cella turcica, etc. (Fig. 160).

Symptoms.—The disease is usually of insidious onset. There is generally noticed at first an enlargement of the hands, feet, and face; the fingers become thickened and swollen, and the entire hand becomes thicker and larger than normal, this enlargement involving both the bones and the soft parts. In some cases the bones of the wrist also become affected. Simul-

Fig. 161.—Acromegaly—front and side view of face and hands.

taneously the feet show a similar enlargement. The enlargement of the hands may bring about either of two characteristic appearances—the hand may become broad, the fingers not being elongated; or, in addition to the increased width of the hand, the fingers become very much elongated (Fig. 161).

The long bones of the extremities are not markedly involved, so that the hands and feet are out of proportion to the remainder of the extremities. The skin is thick and hard, does not pit on pressure, and is usually moist. The openings of the sebaceous and sweat-follicles become enlarged. The head may also become affected; generally the circumference of the skull is in-

creased, and the external occipital protuberance becomes strikingly prominent. The bones of the face are particularly affected; the condition of the lower jaw is characteristic: it becomes enlarged and thickened, and projects beyond the upper jaw, especially in the midline (prognathism); the entire face becomes elongated, marked thickening taking place in the frontal bones, and giving rise to very prominent orbital ridges and zygomatic arches, etc. The eyelids and lips become thickened: the lower lip is heavy and everted; the tongue is broad and thick, and sometimes of enormous size; the mucous membranes are frequently hypertrophied; the palate and larynx are usually hypertrophied, and the voice is deep and coarse. The vertebral column, clavicles, and sternum are also generally affected. The vertebræ of the inferior cervical and superior dorsal regions become enlarged and thickened, and kyphosis in this region is generally present to a marked degree. The sternum is broad and thickened, and the ensiform process is especially prominent. The ends of the clavicles are enlarged, elevated, and thickened. The ribs and patellæ may also be affected. In some instances the patient's entire body undergoes enlargement, giving rise to a condition in which both gigantism and acromegaly are present. A condition resembling arthritis deformans is sometimes seen.

In some cases the disease is ushered in with symptoms of cerebral tumor; in other cases these occur only late, or not at all. Polyuria and glycosuria are frequently present, either early or at the close of the disease. Menstrual disturbances, headache, may occur early or not until late in the disease, disorders of vision, disturbances of the field of vision, optic atrophy, choked disc, ocular palsies, exophthalmos, trifacial neuralgia, etc., may occur during the course of this condition. Epilepsy has been but rarely observed.

The abdominal and thoracic organs are at times hypertrophied, and in some cases dyspnea, cardiac palpitation, and attacks of syncope may give rise to considerable discomfort. The mammary glands and uterus may be enlarged or atrophied. The male or female genitalia may be enlarged or undego retrogressive changes. Sterility is frequent, and very often in males impotence is present. Pain may or may not be a distressing

symptom; it is generally present, however, and in some cases is of a neuralgic character. Pain in the muscles may occasion great distress, and paresthesias, especially in the hands, are frequently present. The reflexes may be normal, increased, or diminished. The movements of the patient are usually heavy and awkward; as a rule, the individual lacks energy and is drowsy and mentally slow.

Course.—The deformity generally develops slowly. The symptoms referable to tumor of the pituitary gland may be of rapid development or may appear only very slowly. As a rule, the general health of the individual suffers. The disease may run a very protracted course, lasting many years, with periods during which there is an apparent improvement or arrest of the condition. In some few cases the course of this disease may be very much more rapid.

Prognosis.—The disease usually lasts many years, the patient dying of an intercurrent affection. The outlook for the arrest of symptoms seems to be somewhat more favorable if proper operative procedures are instituted early.

Diagnosis.—In advanced cases the diagnosis is made without difficulty; it is only in the early stages, when there is but slight if any enlargement of the bones and soft parts, that the diagnosis presents much difficulty.

In the early stages this disease must be differentiated from *myxedema* and *cretinism;* in these diseases there is a loss of hair, the skin is cold and dry, there is no involvement of the bony parts, and a deposit of peculiar, fat-like substances is seen in the supraclavicular and axillary spaces; in acromegaly the skin is moist, there is no loss of hair, bony changes are present, and there is no deposit of fat-like substance in the neck or in the axillary spaces.

Acromegaly and gigantism are closely related, and a combination of the two conditions takes place in many instances. Both are probably due to the same cause, the one condition developing before adolesence, whereas the other develops after adolescence. In either condition changes that are characteristic of the one disease may occur in the other.

From *elephantiasis* acromegaly is distinguished by the fact

that in the former disease only the skin and cellular tissues are involved, and the disease affects only one side or one entire limb. From *osteitis deformans* the disease is differentiated by the fact that in the former disease the legs are principally affected, whereas the hands and feet are unaffected, and the soft parts show no changes; moreover, this disease generally appears after the age of fifty. From *hypertrophic osteo-arthropathy* (Marie) this disease is differentiated by the following signs:

Osteo-arthropathy	Acromegaly
The increase in size affects mainly the bones.	The increase in size affects both the soft parts and the bones.
The carpometacarpal joints are generally affected.	The carpometacarpal joints are generally unaffected.
The lower jaw is normal; kyphosis, if present, affects the lower segment of the spinal column.	The lower jaw is always involved, and kyphosis affects the cervicodorsal segment of the spinal column.
This condition usually develops in patients suffering from prolonged pulmonary or cardiovascular disease, etc.	These diseases are not, as a rule, present.

Fig. 162.—Hands in osteo-arthropathy.

x-Ray examinations always reveal characteristic deformities of each disease.

Treatment.—As this disease is believed to be due to an oversecretion of the anterior lobe of the pituitary gland, the object of treatment is to bring about a lessened secretion of the gland. The administration of thyroid extract, of atropin, of mercury, and of the iodids may all be tried. Operative procedures for the removal of parts of the gland may be instituted; when undertaken by surgeons who have the required training the operation may be performed with a reasonable degree of safety.

INFANTILISM

Infantilism is a condition that is characterized by the absence of or delayed development of the primary sexual characteristics (that is, a delay or an absence in the development of the genital organs), and an absence of or delayed development of the secondary sexual characteristics (that is, the changes in the body and mind that usually occur as the individual passes from childhood into adult life; for example, the figure loses its childish contour, and the characteristic peculiarities of the male or female appear; the voice changes, there is an increased growth of hair in the pubic and axillary regions, and changes in the individual's emotional and intellectual nature take place).

"Failure of the primary and secondary sexual characteristics to appear at the proper time constitutes infantilism; this is equally so whether the general bodily growth be diminutive, normal, or gigantic."

Etiology.—In most cases the underlying cause exists from birth or early infancy; in some instances the condition begins during later childhood, and in a few cases a retrogression of the sexual characteristics sets in in adult life. Among the possible causes of this condition is a defective development of the vascular system in general. Cases due to this condition are generally alluded to as of the Lorain type, and were described by Lorain in 1871.

Chronic infectious or other diseases, drugs, intoxications, etc., such as chronic tuberculosis, either in the parents or in the patient, congenital syphilis, malaria, pellagra, heart disease, severe nutritional disorders, alcohol, lead, morphin, tobacco,

mercury, etc., which interfere seriously with the general nutrition of the subject, give rise to the cachectic form of infantilism.

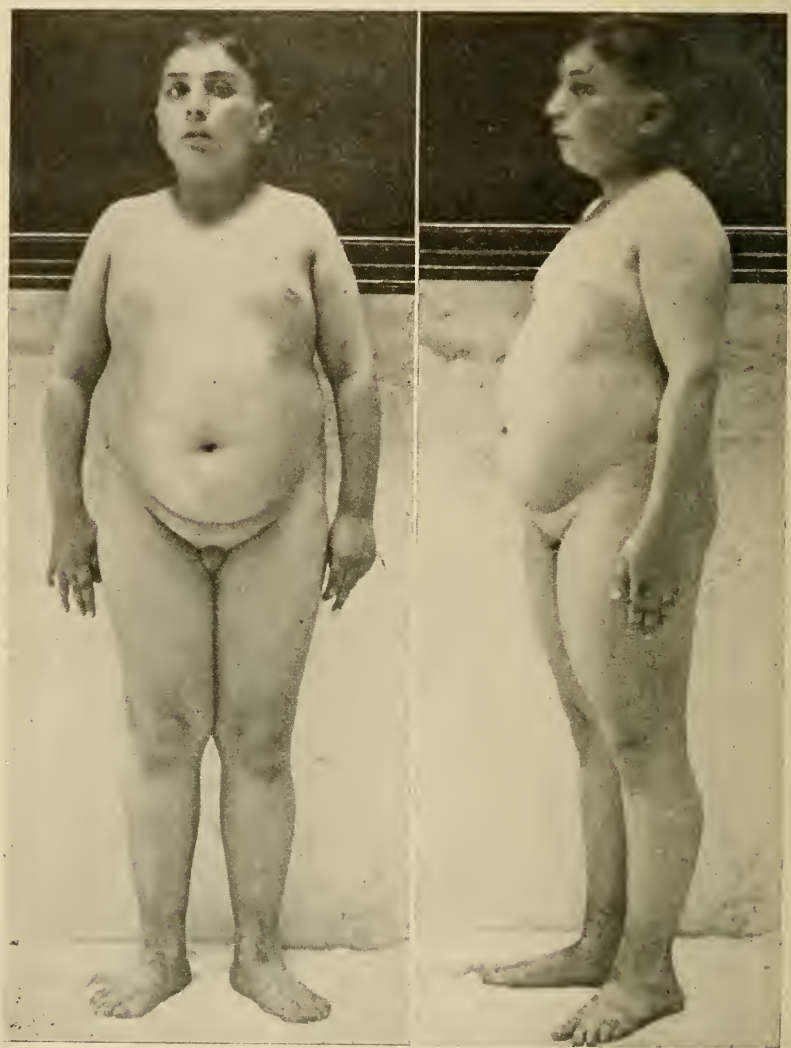


Fig. 163.—Front and side view of a case of infantilism. Notice adiposity and infantile genital development, age twelve years.

There is a third group of causes which may give rise to this condition. These causes are dependent upon gross lesions, defects, or interference with the functions or secretions of im-

portant organs or glands; of these, the most important is deficiency in the secretion of the thyroid gland, the posterior lobe of the pituitary, less commonly of other ductless glands, the pancreas, liver, etc.

Fig. 164.—*x*-Ray of skull in infantilism. Notice abnormally small sella turcica.

Pathology.—As the result of a deficient secretion of these glands, there is a lack of development of the genital organs and an absence of the changes that take place in the body in normal development from infancy to childhood, childhood to puberty,

etc. In infantilism, therefore, the genital organs are very small, the general physical contour is that of a child, hair fails to appear in the axillæ or on the pubes, menstruation is absent, the skin is usually soft, the individual is generally plump, etc. (Figs. 163, 164).

Diminutive size is not necessarily present, as infantilism associated with gigantism is not uncommon; on the other hand, diminutive size with normal primary and secondary sexual development frequently takes place.

Symptoms.—The condition is characterized by the absence of genital development, the genital organs being very small. As the individual grows older, in the female the mammary glands fail to enlarge, hair does not appear in the pubic or axillary regions, and menstruation does not occur; in the male the genitalia remain small, and hair does not appear in the pubic or axillary region or on the face. The intelligence may be fairly normal or remain child-like and immature. There is usually a marked retardation in the bodily development, the physique usually remaining diminutive and resembling that of a young child. There are, of course, exceptions to this condition, and in these there may be abnormal growth of the body, giving rise to gigantism.

In addition to the delayed or absent primary and secondary sexual characteristics, many symptoms occur that are dependent upon the causative condition.

Course.—This condition is usually very slowly progressive. There is noticed in these individuals a delayed and deficient physical, mental, and genital development.

Prognosis.—The prognosis in most cases is rather unfavorable. If due to deficient thyroid secretion, and if treated before adolescence has occurred, the outlook is favorable. If it is due to disease of the pituitary body, the prognosis depends upon the possibility of removing the cause, which is that of pituitary disease in general.

Diagnosis.—It is important to differentiate the various types of infantilism, so that proper treatment can be instituted. This can be done only by going carefully into the history of the case, and making careful examinations to discover the presence

of diseased conditions and abnormal states of the secretions of the ductless glands. It is also important to differentiate infantilism from such conditions as achondroplasia, lack of mental development due to organic cerebral disease or to lack of cerebral development. These latter conditions are differentiated from infantilism by the fact that in them normal development of the sexual organs is generally present.

Treatment.—It is most important, in these cases, to look after the general health of the individual; to treat any of the possible causes that may give rise to a cachectic state. If the condition is due to deficient thyroid secretion, the treatment is the same as that of cretinism; that is, administration of thyroid gland extract, beginning with small doses, and gradually increasing them until the physiologic effect of the drug has been produced, then discontinuing the remedy for a few days, and gradually resuming the treatment with smaller doses.

In cases in which deficient secretion of the pancreas is a probable cause, the effect of the administration of pancreatic extract is to be tried. In those cases in which the pituitary gland is at fault one may try the administration of the extract of this gland. In all cases in which a probable cause cannot be ascertained it is well to administer thyroid extract.

A nutritious diet should be ordered, and the patient should be instructed to drink sufficient water. The bowels should be regulated, and physical exercise be prescribed in accordance with the needs and physical condition of the individual.

DISORDERS OF THE PINEAL GLAND

The functions of the pineal gland have not as yet been definitely determined. There is still quite a diversity of opinion as to whether the gland possesses a definite function or whether it is the remains of an embryonic structure. Most authorities believe that the gland has definite functions that are active during early life, and that, after adolescence, these functions are gradually lost. The gland is believed to secrete a substance that enters the blood and that has an influence on normal growth and on the development of sexual maturity.

Pathologic States of the Pineal Gland.—In addition to local or general cerebral manifestations, diseased conditions of the pineal gland usually give rise to the following symptoms: An abnormal rate of growth, with a premature sexual maturity;

Fig. 165.—Case of pineal gland syndrome, boy thirteen years old, 5 feet 6¾ inches in height. Notice adult genital developement, etc. This patient has had epileptiform seizures since second year.

the increase in size is symmetric, and corresponds to that which occurs under normal conditions in an older individual; *i. e.*, as if normal physical and sexual maturity were attained with an abnormal degree of rapidity and at a prematurely early age (Figs. 165 and 166).

Evidence of *abnormal size* and premature sexual maturity appears at a very early age. Epilepsy occurs in many cases. The intellectual development may be normal for the age or retarded; it is very rarely above normal.

The course is usually slow, the first evidence appearing within the first few years of life. The symptoms are slowly progressive,

Fig. 166.—*x*-Ray showing normal sella turcica in patient shown in Fig. 165.

the patient dying either of some intercurrent disease or as the result of the local pathologic condition bringing about a disturbance of intracranial pressure and subsequent death.

The prognosis is extremely grave. In most cases the condition is gradually progressive, death occurring before the twentieth year; in some cases it occurs during the first few years of life.

If the condition is due to a tumor in the neighborhood of the pineal gland, operative procedures may possibly avert a fatal outcome, but the operation itself is very difficult, and, except in the hands of the most expert, is usually followed by a fatal outcome.

The diagnosis is made from the presence of an abnormal rate of growth, unattended by abnormalities of bone and muscle development. The increase in stature is such as would be normally present in a much older individual. There is a premature development of the sexual organs and of the sexual functions. Mentally the individual is not advanced beyond his age, and often there is evidence of a mental deficiency. In addition to these symptoms there may be evidences of a cerebral tumor or of internal hydrocephalus. The *x*-ray may be of help, especially in diagnosing calcareous deposits that have taken place in the neighborhood of the pineal gland. The *x*-ray fails to reveal abnormalities in the sella turcica, the bones of the skull and face, and the bones of the extremities, such as occur in gigantism with precocious sexual development (Fig. 166).

Treatment is wholly surgical. A decompression operation may be performed where there is marked increased intracranial pressure or if choked disc exists, and operative attempts to rectify pathologic conditions of the pineal gland have also been made.

DISEASES DUE TO DISTURBANCES OF THE VASOMOTOR SYSTEM

ACROPARESTHESIA

Etiology.—Exposure to cold, the frequent and prolonged immergence of the hands in cold or in hot water, overuse of the hands, etc., are among the common causes of this condition. The disease occurs more frequently in women than in men. It usually occurs after the thirtieth year, but may appear earlier. It is more apt to affect washerwomen, cooks, seamstresses, etc. Poor hygienic surroundings, anemia, chronic intoxication, injury, grip, etc., have also been mentioned as causes of this condition.

Pathology.—This affection is probably due to an interference with nutrition or to an irritation of the sensory nerve-endings in the extremities, caused by vasomotor disturbances. Such disturbances are either due to local conditions, such as prolonged exposure to cold or to hot or cold water, toxic conditions, etc., or are of central origin, the result of overwork.

Symptoms.—The individual usually complains of paresthesia in the fingers, sometimes extending to the hands. There is a feeling of burning and numbness, which is most severe in the tips of the fingers. In some cases the patients complain of a feeling of burning; others of itching, and some of coldness and stiffness of the fingers. Pain may be complained of, but is usually not severe.

The paresthesias are more or less constantly present, but they vary in degree. As a rule, they are more marked in the mornings, immediately after arising, improve somewhat during the day, but are usually intensified upon exercise or exposure. A careful objective examination of these individuals may show some slight decrease of sensibility in the finger-tips, but even this is

absent in many cases. Many of these patients are of a neurotic disposition.

Course.—The affection usually develops very slowly and runs a chronic course. It may last for many years.

Prognosis.—Under proper treatment a complete disappearance of the symptoms usually occurs. Some cases are very resistant to treatment and persist for years. Relapses are very apt to follow undue exposure.

Diagnosis.—A careful examination should always be made to exclude other diseases that may begin with the paresthesia; the more important of these are tabes dorsalis, organic disease of the cervical enlargement of the spinal cord, Raynaud's disease, ergotism, tetany, hysteria, multiple neuritis, severe anemias, etc.

Treatment.—It is most important to remove the cause. Overuse of the hands and too free use of cold and hot water, etc., should be avoided. The individual's general health should be carefully looked after, and tonics containing iron, strychnin, arsenic, etc., should be given. The daily local application of the faradic or of the faradic and galvanic current seems to do much good.

MIGRAINE (HEMICRANIA)

This disease is characterized by sudden attacks of intense pain on one side of the head, which are frequently preceded by signs of irritation of the various functions of the brain. The pain gradually becomes worse, until, when the greatest intensity is reached, there occur digestive disturbances, with more or less prostration, followed by gradual relief.

Etiology.—Heredity is the most important factor. According to Möbius, in 90 per cent. of cases the disease is directly inherited. Prolonged emotional disturbance, debilitating conditions, excitement, overwork under unfavorable conditions, etc., may predispose or bring on an attack. The disease may also be of reflex origin: refractive errors, diseases of the generative organs, throat, nose, and accessory sinuses, or of the abdominal organs, etc., may either predispose to the disease or bring on an attack.

The disease manifests itself for the first time usually during early childhood; rarely does a first attack occur after the eighteenth year. The exciting causes of individual attacks very often include excitement, variations in the atmospheric pressure, excesses of any kind, such as overindulgence in eating or drinking, or overwork, mental or physical.

Pathology.—A careful examination fails to reveal any constant pathologic change either of the central nervous system or in the body. Two theories are held regarding the causation of the attacks. One is that there is produced in the body, either as the result of excitement or of some other disturbance, a toxic substance that at first produces stimulation of the brain, causing a sense of well-being and of increased capacity for work and exhilaration. This is soon followed by a secondary and more lasting effect, which evidences itself as pain and depression of the cerebral functions; further, as the result of the elimination of this poison through the stomach and intestines, gastro-intestinal symptoms occur, a normal state of health being again attained after the poison has been eliminated. The nature of the toxin is still undetermined. The other theory is that this disease is of vasomotor origin, and that the symptoms are due to a temporary localized interference with the cerebral circulation. It would appear as if both of these theories were correct: that this disease occurs only in individuals who, as the result of a hereditary taint, are either susceptible to certain poisons or in whom there is a tendency to the formation of specific toxins, which at first cause a vasomotor dilatation, this being followed in turn by a vasomotor spasm. The vasomotor disturbances are more marked on one side of the brain than upon the other. The prostration is due either to the effect of the toxemia or to the exhaustion of the vasomotor centers, which have been overstimulated; the gastro-intestinal symptoms are the result of nature's efforts to eliminate the toxic substances.

Symptoms.—In many cases the attack is preceded by a period of excitement or by a feeling of well-being which may last for several hours and sometimes for several days. In other cases the attack is preceded by a feeling of exhaustion, drowsi-

ness, fulness in the head, depression, etc. Again there is another group of cases in which ocular migraine is generally preceded

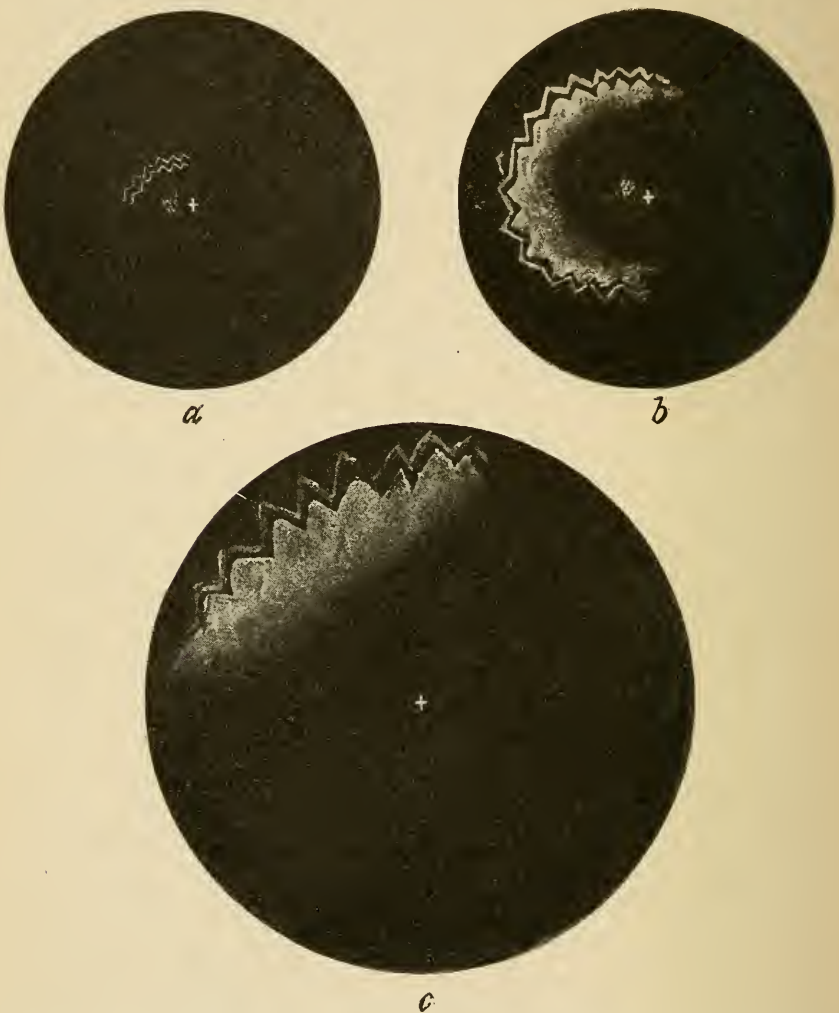


Fig. 167.—*a*, *b*, and *c* represent the ocular phenomena in the field of vision at different stages during an attack of migraine, when the eyes are closed. The central white cross is the point to which the sight was directed; the zigzag lines and diffused coloration represent the scintillating scotoma (Mackinzie, in Allbutt's "System of Medicine").

for from a few minutes to a half-hour by the appearance of flashes of light, by bright spots, which may be of any color,

before one or both eyes, and which sometimes gradually enlarge to form zigzag lines or fortification spectra (Fig. 167). These are true visual hallucinations, as they are continued in the dark or on closing the eyes. These attacks may also be preceded by a partial or complete hemianopsia, and more rarely by blindness.

Either with or without these premonitory signs the characteristic symptom of the disease—headache—occurs. The headache does not, as a rule, commence in its full intensity; it is usually dull at first and gradually grows worse until it becomes almost unbearable. It is not generally limited to one side, as the name of the disease would lead one to suppose, but frequently affects the forehead and both temples. Pain is present above the eyes and in the occipital region, and frequently is more intense at one point, the location of which may vary at different times; thus one side of the head may be affected more severely than the other, and in some patients the attacks alternate from side to side. Very frequently the patient also complains of a hyperesthesia and tenderness of the scalp.

During the attack the patient is abnormally sensitive to sensory impressions. Bright light or noises intensify the pain, and in order to avoid these he remains in a quiet, darkened room. Movement of the head also increases the pain, and consequently the patient keeps as quiet as possible. During the attack the individual feels weak, there is a loss of appetite, and vomiting generally occurs at the height or toward the close of the attack, at which time, also, diarrhea and polyuria may occur. Finally, the pain begins to decrease and the individual falls asleep, usually awaking free from pain and considerably exhausted, but strength rapidly returns.

In some cases vasomotor and pupillary symptoms occur: the face may become pale, the skin cold, the pupils dilated, and the temporal artery contracted. In other cases the face is red, the conjunctivæ are injected, the temporal artery is dilated and pulsating, and the pupils are contracted. In some few cases speech disturbances (such as incomplete aphasia), paresthesia, or weakness, limited to one limb or to the side of the body opposite to that on which the most pain is experienced, occur.

In some subjects the attacks are limited to pain in the head, prostration, and gastro-intestinal symptoms; in others these symptoms are accompanied by signs of irritation or by depression of localized cerebral areas. The intensity of the individual attack may vary. No definite periodicity of these attacks is seen—they recur at irregular intervals, especially after excitement or following mental or physical indiscretions; in women they may occur every three or four weeks, in these cases bearing some relation to menstruation. Occasionally the attacks may be very mild and easily aborted by medicine, occupation, etc.

Course.—The disease usually manifests itself in early childhood and recurs in attacks at irregular intervals for many years. In some cases there is a tendency for the disease to disappear in later adult life.

In many cases when the individual has passed middle life, if complete relief does not occur, the vomiting and the ocular symptoms disappear. This disease is often associated with neurasthenia and hysteria, and symptoms of these neuroses are often present during the intervals between the attacks.

Prognosis.—The prognosis in each individual attack is, of course, good, there being practically no danger to life, although the prostration may be extreme. The attacks usually last for from twenty-four to seventy-two hours, rarely longer.

With proper treatment and regulation of the patient's life the intervals between attacks can be greatly lengthened, and in some cases the attacks entirely prevented. In most cases the migraine appears at irregular intervals, finally becoming less severe, and often disappearing entirely after the fiftieth year. Attacks also disappear during pregnancy, and sometimes when the patient changes his mode of living, or lives amid different climatic conditions, changes his occupation, etc. These individuals usually become prematurely aged and gray. In a very small number of cases the migraine attacks cease and epilepsy develops.

Diagnosis.—The diagnosis of this condition, as a rule, presents very little difficulty. It must be differentiated from hysteria, cerebral tumor, meningitis, toxemias, etc. Any difficulty that may arise is present only in the first attacks. The condition

is differentiated from *hysteria* by the fact that in the latter disease the individual usually does not wish to be alone; the pain is rarely limited to the head; the vomiting occurs early, and the patient is amenable to suggestion; in migraine, on the other hand, the individual wishes to be left alone, because light, sound, and movement tend to increase the pain; he appears weak, and the vomiting occurs toward the close of the attack.

In *cerebral tumor* the pain is more or less constantly present, the pulse is usually slow, and ophthalmoscopic examination frequently shows fundus changes; irritative symptoms, when present, persist for a time, and then, as succeeding portions of the brain become affected, gradually merge into paralytic symptoms.

From *meningitis* the condition is differentiated by the physical signs of meningitis and the examination of the cerebrospinal fluid. From *cerebral syphilis* migraine is differentiated by the history, the examination of the blood and the spinal fluid, and the effect of the treatment; from *toxemias*, either of gastro-intestinal or of nephritic origin, a careful physical and clinical examination should render the diagnosis comparatively certain. When the attacks have recurred at irregular intervals for any length of time, particularly in the presence of a history of a similar condition in the ancestry, the diagnosis is comparatively simple. It should be remembered that individuals who are subject to migraine may also acquire organic diseases, and therefore whenever an attack occurs that is accompanied by symptoms of an unusual nature, or that is unduly persistent, a careful examination should be made to determine that no other condition has developed in addition to the migraine.

Treatment.—The treatment of this condition should be directed toward improving the general health of the patient, so as to prevent a recurrence of the attacks, or to lengthen the intervals between attacks. Relief of the individual attacks is also indicated.

General Treatment.—The person who is subject to attacks of migraine should lead a simple life, free from excitement, avoiding both mental and physical overexertion; he should follow some

congenial occupation. Undue exposure to either too hot or too cold weather should be avoided, and, if possible, he should live in some mild, stimulating climate; if this is not possible, he should so regulate his attire as to meet weather conditions. The diet should be light, nutritious, and non-stimulating; all food should be thoroughly masticated before it is swallowed, and it is better for the patient to eat four small meals a day than two or three large meals. Heavy pastries, rich foods, and alcoholic drinks are best avoided. The patient should drink at least two quarts of water a day. The bowels should be regulated, and under no condition should constipation be allowed to occur. Exercise in moderation is to be encouraged, but physical fatigue or sports in which excitement plays a part should be avoided. The patient should spend as much time as possible out-of-doors, and should rest for a short time after each meal.

Bathing.—Hot alkaline baths, followed by cold sponges, hot baths, or Turkish baths once a week, are all beneficial. The Scotch douche, taken each morning upon arising, is also helpful in improving the general tone of the patient. A course of treatment at Carlsbad is helpful in many cases. Massage, properly given, is of much service. Physical defects, such as errors of refraction, diseases of the throat and nose, and abdominal and pelvic disorders should receive proper treatment, if possible, corrected. If anemic, patients should be given preparations of iron and arsenic, and in many instances it is well also to give strychnin in proper doses. Starr has found the administration of thyroid extract of considerable service. Bromids given in small doses are often of advantage to those of a nervous temperament.

Treatment of the Attack.—In the milder cases it is occasionally possible to abort the attacks by washing out the stomach with an alkaline solution; in other cases this may be done by the administration of a saline purgative, and at times by the application of an ice-cap or of a hot-water bottle to the head, and also by the prompt administration of drugs, such as a large dose of the bromids, or a capsule containing aspirin, 5 grains; caffein citrate, 1 grain; camphor monobromate, $\frac{1}{2}$ grain; and codein sulphate $\frac{1}{4}$ grain; this capsule may be repeated within

one-half hour, and then every two hours, until four or five capsules in all have been taken. At times the patient will cease to respond to aspirin, but will react to phenacetin, pyramidon, etc. In some patients a cup of hot strong coffee will abort the attack; again some individuals obtain relief from the administration of $\frac{1}{100}$ grain of nitroglycerin. If these procedures are not successful in aborting the attack, the patient should be placed in a quiet, darkened room, one that is well ventilated and kept cool. It is well to apply ice-caps to the head and hot-water bottles to the feet. The nausea may be relieved by mustard plasters or other counterirritants applied to the epigastric region. In the early stages the coal-tar products, combined with codein, caffein, camphor monobromate, nitroglycerin, strychnin, etc., will lessen the pain. When the nausea and vomiting become intense, nothing is retained by the stomach. During the attack it is advisable to abstain from all food. Liquids should be given at frequent intervals in small quantities. It is also well early in the attack to give a saline purgative if this can be retained; if this is impossible, calomel may be tried; this may be given in one single dose of 2 grains, or in eight doses of $\frac{1}{4}$ grain each, given at intervals of fifteen minutes. If the lower bowel contains much fecal matter, a hot soapsuds enema should be given.

In those cases that are not benefited by any of the foregoing measures a hypodermic injection of morphin may be given; this should, however, be used only under exceptional conditions, as there is great danger in these cases of creating the morphin habit; moreover, the administration of morphin certainly exerts an unfavorable effect on metabolism, thereby predisposing the patient to more frequent attacks. Most patients sooner or later succeed in finding some drug or some procedure that affords them relief; or, finding that nothing seems to give relief, they refuse all medicines during the attack, preferring to remain quietly in a darkened room, taking only a small amount of liquid nourishment, and bearing the pain with patience until the attack has passed off.

ERYTHROMELALGIA

Erythromelalgia, or red neuralgia, is a condition that was first described by Wier Mitchell. It is characterized by pain, swelling and redness of the skin of the distal parts of the feet, of the hands, or of all four extremities.

Etiology.—This is a somewhat uncommon affection. Men are more frequently affected than women. Exposure to cold seems to be the principal predisposing cause. Nothing definite is known as to the true cause of this condition.

Pathology.—The disease has been described as an angioneurosis; as an affection of the vasomotor centers; as a diseased condition of the spinal cord. In one case Wier Mitchell found marked degeneration of the fine nerve branches of the peripheral nerves. The most constant findings are pathologic states of the smaller peripheral arteries. In the opinion of Barlow, Buerger, and others, in erythromelalgia we have a disease in which there is an affection of the smaller vessels of the extremities, resulting in disturbance of circulation—in other words, a condition that is closely related to intermittent claudication. The alternating states of coldness and hyperemia, pallor, blueness, and redness, may be explained by varying degrees of spasms and dilatations of the arteries that have to do with the collateral circulation. The pain in these conditions may be due to the spasms of the arteries themselves, to disturbances of the circulation of the nerves, or to muscular cramps; to what extent any one of these factors enters into the production of pain it is difficult to say.

Symptoms.—Pain is the most marked symptom. This may come on suddenly, and sometimes follows overexertion. The pain occurs in the feet, especially in the balls of the toes and in the heels, or in the hands and fingers, or in all four extremities. The pain is accompanied by redness, which is most marked in the distal portions of the affected extremities. With the redness there is usually slight swelling. The patient complains of a feeling of heat. The blood-vessels generally pulsate, and there is a slight objective rise of the skin temperature, and frequently a local hyperidrosis occurs. Sensibility is not, as a rule, affected. The redness, swelling, and pain vary in intensity. The pain is

generally severe, and may increase to such an extent as to be practically unbearable; it is somewhat relieved by elevating the extremities. Cold applications also afford some relief from pain, whereas heat increases it. In some cases the congestive appearance becomes so marked that the hands or the feet, or the hands and the feet, take on a dark purplish color. In addition to the local symptoms there may be headache, vertigo, general weakness, and cardiac complications.

A careful physical examination usually fails to reveal any causative condition.

Course and Prognosis.—There is no treatment known that seems to affect the course of the condition. The disease may begin suddenly or gradually and remain more or less constantly present; at times there are temporary remissions. The condition is a very distressing one, and the pain may become so severe that there is danger of the patient attempting suicide.

Treatment.—Cold applications and elevation of the affected parts seem to afford some relief. Operations for the removal of the nerves are at times resorted to, and in some instances amputation has been necessary. The various drugs for the relief of pain may be used. It may be necessary at times to resort to general anesthesia in order to afford relief, and careful supervision of the patient be necessary to prevent suicide.

RAYNAUD'S DISEASE (SYMMETRIC GANGRENE)

This disease is characterized by hyperemia or ischemia, occurring and terminating more or less suddenly, in symmetric parts of the body, especially in the phalanges of all the extremities. It may or may not result in trophic changes or in gangrene.

Etiology.—Heredity, as evidenced by the occurrence of neuroses in the ancestry and of a neuropathic disposition, is present in many cases.

The disease occurs most frequently between the fifteenth and the thirtieth year, but it may occur in very young children and in older persons. Females are more prone to be affected than males.

Anemia, exhaustion, trauma, overexercise, infectious diseases,

such as influenza, typhoid, erysipelas, pneumonia, etc., chronic diseases of the kidneys, heart, and blood-vessels, chronic poisoning by lead and alcohol, diseases of the genital organs, exposure to cold, and emotional disturbances, are all believed to be among the predisposing causes. Certain occupations seem to predispose to the condition; thus washerwomen, waitresses, and chambermaids constitute the class most often affected. Marked emotional disturbances seem to occur very frequently before the onset of the disease. Many cases are seen in which it is impossible to discover a probable cause.

A symmetric gangrene may also occur during the course of certain nervous diseases, such as hysteria, tabes dorsalis, syringomyelia, multiple sclerosis, tumors of the spinal cord, epilepsy, exophthalmic goiter, traumatic neuroses, dementia præcox, myelomalacia, etc.

Pathology.—The nature of the disease is still undetermined. Some investigators, among them Raynaud, held that this disease was of central origin, the dilatations and contractions of the vessels being due to an abnormal process going on in the vasomotor centers in the spinal cord. Other investigators believe that the vasomotor conditions are the result of a reflex irritation arising in the periphery or brain, this irritation affecting the vasomotor centers and thus bringing about the vascular disturbances. Still others claim that the primary disease affects the blood-vessel walls and the nerve-endings in the vessels. As the result of some pathologic process there develops a condition of the arterioles that may vary in degree from partial to complete occlusion, resulting in paleness or cyanosis, which may be of temporary duration, or which may be followed by gangrene of the affected part, depending upon the extent and the duration of the interference with the blood-supply. In other cases spasm of the smaller veins may occur, bringing about redness, which may be followed in turn by gangrene. The spasm of the vessels may begin and end suddenly.

Symptoms.—In practically all cases in which the manifestations of this disease occur independently of other nervous affections, the onset is sudden. In some few cases the local symptoms are preceded by general constitutional disturbances,

the individual becoming irritable, restless, and depressed. Headache, loss of appetite, and gastro-intestinal disturbances may occur; then, more or less suddenly, the local symptoms develop. The attack usually commences with paresthesia, a feeling of numbness or deadness, a burning or tingling, which finally, as a rule, develops into a marked painful state of the fingers and toes. The paresthesia and the pain may be present for days or weeks preceding the attack, and may increase with its onset.

Hyperesthesia or hypo-esthesia may be present during the early stages of the attack. Either following or preceding the paresthesia and pain there is an ischemia of the affected extremity. The further course of the attack depends upon the extent and duration of the vascular occlusion; in the first stages the fingers and toes become white and cold, like those of a dead person, and the prick of a pin is not followed by bleeding. Along with this condition there is usually violent pain. In some cases the interference with the circulation is not quite so complete; there is, as a rule, some impairment of sensation, the finer movements become clumsy, and actual anesthesia may exist. The affected parts become shrunken, and the skin is thrown into folds and is somewhat hard and immovable. The secretion of sweat may be increased or entirely suspended, and the local temperature is lowered. The duration of the attack varies from a few moments to several days. The spasm may relax suddenly, and the part return to the normal condition, the return of the blood, as a rule, being accompanied by a burning sensation. This first stage may be followed by a cyanosis, or the cyanosis may exist from the onset. The cyanosis results either from a complete arterial spasm, in which case the venous blood regurgitates into the capillaries and distends them, or, as the result of a venous spasm, there occurs a congestion of the part, the blood in the capillaries finally becoming venous and thus producing the cyanotic appearance. In some cases the stage of ischemia is so short as to pass unnoticed, and the patient's attention is first attracted by the swollen, blue, and painful condition of the affected part.

Stage of Cyanosis.—The affected parts appear blue and swollen,

are frequently infiltrated, feel cool, and are generally hyperesthetic. Severe pain is present. The parts can usually be moved. When this condition persists the parts become blue-black and finally black, and the epidermis may show vesicles and slight hemorrhages. Even with this condition there may be a sudden

Fig. 168.—Raynaud's disease, showing spontaneous amputation of fingers and toes; areas of gangrene (case of Dr. Arthur Shipley).

relaxation of the spasm and the parts resume their normal appearance, although, as a rule, this is slower than after simple ischemia. This second stage may last from a few seconds to several days, and is usually followed by gangrene.

The third stage, or stage of gangrene, is always preceded by cyanosis. In the cyanotic areas small blisters filled with bloody serum appear; these soon rupture, then dry, and dark crusts form, beneath which ulceration takes place. In some cases the gangrene is limited to the superficial tissues and the ulcer heals over. In other cases the ulceration may involve the deeper structures, which may gradually granulate and heal. In still other cases the entire skin of a terminal phalanx may become black and dry and undergo gangrene. Gangrenous involvement of an entire phalanx is the exception rather than the rule. The dead tissue is bounded by a line of demarcation. With the completion of the stage of gangrene the local symptoms terminate. It is the symmetric distribution of the local symptoms which is characteristic of the disease; thus the fingers of both hands, toes of both feet, more or less symmetrically situated spots upon the forearms, arms, legs, thighs, and trunk are affected, singly or in combination (Fig. 168). In many cases the fingers and toes are chiefly affected, only a few spots occurring on the body; very often the toes escape. The face is frequently affected, spots of cyanosis appearing on the nose, ears, or lips.

As a rule, the gangrene affects only the tips of the extremities and small areas of skin on the forearm or arm. In addition to the characteristic symptoms of symmetric gangrene, other manifestations occur: in some cases there is atrophy of the small muscles of the hand, or trophic disturbances of the nails and skin occur. Trophic disturbances, with swelling, redness, and effusion of the joints; visual disorders attributed to spasms of the retinal arteries; deafness, tinnitus aurium, and disturbance of the sense of taste, etc., may occur as transient symptoms. Oculomotor and pupillary disturbances, temporary albuminuria, glycosura, transient aphasia, epilepsy, and paralysis of cerebral origin may also occur. Mental disorders, such as depression, excitement, confusion, etc., may precede or accompany the attack.

Course.—The onset of an attack is usually sudden. The symptoms of the first two stages, that is, the symptoms of ischemia and of the stage of cyanosis, may last for only a few minutes and pass off, or the first stage last a short time, the

second stage longer, and then the symptoms disappear; or the second stage may be long or short and terminate in gangrene.

As a rule, the first and second stages last several days, and the stage of gangrene continues for several weeks. In some individuals the disease is limited to a single attack; in others recurrences occur within a year, the disease lasting, with remissions, for several years, and in some cases for many years.

If the gangrene begins simultaneously in all the affected parts, the duration will be shorter than if it extends from one part to another. The attacks may rapidly succeed each other, or there may be a prolonged interval between attacks. Some attacks are very much more severe than others. Moderate vasomotor disorders are usually present during the intervals between the attacks.

Prognosis.—It is impossible to state with certainty whether or not a recurrence of the attacks will take place, nor can one state with positiveness that recovery from the individual attack is to be expected. The duration of the attack depends upon the character, the extent, and the severity of the symptoms. Life is not endangered by this disease.

Diagnosis.—Raynaud's disease must be differentiated from *frost-bite*. In frost-bite there is usually a history of exposure together with itching and burning that are persistent: in Raynaud's disease the attack occurs suddenly without exposure, and usually pain, more or less marked, is present, which ceases when the vascular spasms subside. From *ergot poisoning* Raynaud's disease is to be differentiated by the history. From *gangrene occurring in hysteric and neuropathic individuals* Raynaud's disease is differentiated by the presence of other symptoms of the hysteric and neuropathic states, and the lack of symmetry in the distribution of the gangrenous areas. From *gangrene occurring in diabetes, in nephritis, in old age, in endarteritis, in spinal cord diseases, in leprosy*, etc., Raynaud's disease is to be differentiated by the history and by careful examination.

The diagnosis of Raynaud's disease rests practically upon the development of the vasomotor symptoms in the extremities or on the trunk, situated in symmetrically situated areas, the symptoms going on to gangrene. The condition occurs usually

in individuals between the fifteenth and the thirtieth year, and is accompanied by pain, which is more or less severe, but which passes off as the arterial spasm relaxes. In Raynaud's disease there is no history of exposure to cold or to ergot-poisoning, and there is an absence of endarteritis, nephritis, diabetes, spinal cord disease, leprosy, etc.

Treatment.—The general health of the patient must be looked after. Tonics and proper hydrotherapeutic measures are indicated. The bowels should be kept well open, the diet regulated, and excitement, worry, exposure to cold, and indulgence in alcoholic drinks are to be avoided. Between the attacks the treatment should be directed to the general building up of the body and of the nervous system. During an attack if the pain is very severe it should be relieved by codein or morphin. If restlessness is marked, the bromids or the bromids and chloral should be given. The patient should be kept quiet, and the affected parts should be protected by enveloping them in soft bandages or cotton. In no case should irritating ointments or the faradic current be used on these parts.

During the attack gentle massage, dry heat, or lukewarm hand- and foot-baths may give relief. Galvanism to the spine has often been tried and is to be recommended, but the faradic current should never be used. A moderately weak galvanic current may be tried, placing the positive pole over the cervical region and the negative pole over the lumbar region; the treatment should last for about ten minutes and should be employed once daily; the current should not be strong enough to cause much discomfort. The galvanic current may also be used on the affected extremities, the positive pole being placed over the spinal column, and the negative pole being gently moved backward and forward over the extremities, care being taken to use weak currents and to turn the current on and off gradually. It has also been recommended that the affected limbs be kept warm by covering them with cotton and bandages, and placing them at rest in an elevated position. If gangrene occurs, the parts are to be kept surgically clean. All local injury and counterirritation are to be carefully avoided.

Residence in a warm climate, such as southern California,

Florida, or Cuba, will generally prevent a recurrence of attacks and rapidly cause a disappearance of all symptoms.

ANGIONEUROTIC EDEMA (ACUTE CIRCUMSCRIBED EDEMA)

Etiology.—This condition generally affects persons of a nervous disposition. It often occurs in combination with hysteria, neurasthenia, and urticaria. Various intoxications, especially of intestinal origin, exposure to cold, excitement, emotional disturbances, and injury are regarded as exciting causes. Nervous or toxic disturbances occurring at the menstrual period may bring on the condition.

It generally occurs in young adults, but not uncommonly affects children.

Pathology.—The nature of the condition has as yet not been established. A local venous spasm or local secretion of lymph has been regarded as a probable cause.

Symptoms.—Attacks of acute circumscribed edema may come on with or without preceding emotional disturbance, exposure to cold, etc. There usually occurs, more or less suddenly, a circumscribed edematous swelling of the skin and sometimes of the mucous membrane. These swellings are rounded or may be irregular in outline; they may develop in different parts of the body or affect one entire extremity, one side of the face, etc. The swellings stand out prominently on the skin, and are either much redder or much paler than the surrounding skin. The temperature of these swellings is somewhat lower than that of the surrounding tissues.

The condition appears to be of the nature of an edema of the superficial tissues. The swelling is hard, does not pit on pressure, and cannot be completely removed by pressure or manipulation. These swellings are usually accompanied by a sensation of stiffness. If they occur about the eyelids, the mouth, or the extremities, motion is somewhat impeded. Burning, numbness, and even actual pain at times accompany these swellings. The discomfort is usually increased by the application of cold. The swellings generally occur in paroxysms, which come on more or less suddenly and last for from a few hours to several days.

In the majority of cases there is a complete cessation of the symptoms between the attacks. The tongue, lips, larynx, pharynx, conjunctiva, cheeks, joints, gastro-intestinal tract, kidneys, etc., may be the seat of the edema. As a rule, the general health is not affected, but the swelling of the mucous membrane may give rise to troublesome symptoms. In individuals suffering from this condition periodic attacks of vomiting, gastro-intestinal pain, asthma, interference with breathing, etc., may occur.

Course.—The disease usually begins suddenly in the form of paroxysms, which may occur frequently or only at intervals. It generally runs a prolonged course; relapses are very common.

Prognosis.—Life is seldom in danger. Recovery from individual attacks is to be expected, and usually, after a considerable period, there may be complete freedom from further attacks. In many instances the condition lasts for years.

Diagnosis.—This condition must be differentiated from *urticaria*. This can usually be done by a careful consideration of the history of the case, and by the fact that in urticaria the lesions are much smaller and do not occur simultaneously, but successively. An intense burning and itching is present in urticaria and absent in acute edema; furthermore, cold applications usually afford relief in urticaria, but aggravate the condition in acute edema.

From *chronic hereditary trophic edema* and from *permanent edema* the condition is differentiated by the fact that in these diseases the swelling is present more or less permanently, and the edema is due to a true hypertrophy of the tissues.

Treatment.—The most important factor in the treatment consists in improving the general health of the patient and building up the nervous system by proper exercise, food, hydrotherapy, rest, massage, and mental training. The diet should be regulated, and the bowels kept well open. Care should be taken to avoid undue exposure to cold and emotional disturbances.

Internally, arsenic, atropin, quinin, and strychnin have all been used with advantage. If one remedy fails, another should be tried. Irritating or cold lotions and applications should be carefully avoided.

TROPHONEUROSES

PROGRESSIVE FACIAL HEMIATROPHY

This condition is characterized by a progressive decrease in size of one side of the face, the atrophy involving both the soft and the hard tissues. Along with the atrophy there is a marked pigmentation of the skin.

Etiology.—This disease is very rare, and occurs most frequently between the tenth and the twentieth year. Injury of the face or skull, operations upon the throat, infectious diseases, such as diphtheria, erysipelas, measles, pneumonia, and a congenital predisposition are believed to be among the causative agents.

Pathology.—By some this disease is attributed to a pathologic condition originating in the Gasserian ganglion or in the sympathetic system, the atrophy being regarded as a secondary trophic condition; other authorities consider this to be a primary atrophy of the cutaneous and subcutaneous cellular tissue.

Symptoms.—This disease generally begins with the appearance of atrophy in one or more parts, especially in the orbital region and the lower jaw. There is a discoloration of the skin, which gradually becomes glossy and undergoes atrophic changes. This is followed by a wasting of the subcutaneous tissues and the disappearance of the fatty tissue. The skin, which may be pale, brown, yellow, or bluish in color, falls into folds, and finally lies close to the bones; gradually the bony structures become affected and decrease in size. The process, which usually starts in one or two places, may remain limited to these parts for some time, or they may gradually extend until all the tissues upon one side of the face are involved; eventually, as the result of this process, there is a decrease in size in every part of one side of the

face; the bones in particular become smaller, and the asymmetry is very evident. The eye is usually deeply sunken, and the cheek furrowed and grooved. In the advanced stage the difference between the two sides of the face is very striking; the atrophy affects the skin, subcutaneous tissues, bones, and sometimes the muscles of the face and tongue. Occasionally the larynx has been involved. The hair is rarely affected, for the atrophy seldom extends to the scalp. Occasionally the atrophy extends to the other side of the face, and may even involve the shoulder, girdle, or one entire side of the body. Sensory disturbances are uncommon. Herpes may exist. The secretion of the sebaceous glands is diminished; the perspiration may be increased; the temperature of the skin is normal.

Electric examination of the muscles shows no reaction of degeneration. Paralysis does not occur, although weakness may exist. The teeth and the eyebrows are usually involved and may fall out.

Course.—This disease is slowly progressive, but sooner or later comes to a standstill. In association with this condition it is not uncommon to observe neuralgia, tabes dorsalis, epilepsy, facial tic, disseminated sclerosis, psychosis, chorea, palsy of the ocular muscles, pupillary disturbances, etc.

Prognosis.—The disease is usually slowly progressive, in rare instances extending to the opposite side of the face or involving the neck, shoulder, or the entire side of the body. As a rule, spontaneous arrest of the condition takes place.

Treatment.—The general health of the patient should be looked after, and careful examination made for any irritative or destructive disease of the vascular system, the sympathetic, or of the Gasserian ganglion or its branches. If any such condition is present, an attempt should be made to correct the same.

Local disease of the throat and nose should receive proper treatment. Local massage of the affected side of the face should be conscientiously used. Tonics containing strychnin should be administered. In those cases in which marked deformity exists, the symmetry of the face may be somewhat, if not completely, restored by the subcutaneous injection of paraffin, as recommended by Gersuny and others.

SCLERODERMA

This disease is characterized by an abnormal condition of the skin and subcutaneous tissues.

Etiology.—Little is known concerning the etiology. Heredity, prolonged emotional disturbances, exposure to cold, a marked rheumatic tendency, injury, etc., have all been known to precede this condition. It has also been observed in paresis, tabes dorsalis, and chronic spinal disease. The disease may occur at any age, but is most common between the twentieth and the fortieth year. Women are more frequently affected than men.

Pathology.—Almost all authorities regard scleroderma as a trophoneurosis. The condition has also been attributed to poisoning or disturbance of the internal secretions.

Pathologic examination of the nervous system has revealed nothing definite. The disease is characterized by a sclerosis affecting the skin and subcutaneous tissues, which is followed by atrophy and contraction. In addition to the skin changes degenerative alterations in the blood-vessels are usually present.

Symptoms.—For some time before the occurrence of the sclerodermic process vasomotor symptoms may occur, such, for example, as local cyanosis, redness and edema, and, at the same time, pain, itching, tingling, and burning. Either with or without these symptoms changes indicative of the disease take place.

The process may be circumscribed, and take the form of spots, or may be diffuse. The spots, which at first are isolated or disseminated, as a rule become confluent later in the disease.

Frequently some part of the body that has been exposed to pressure serves as the starting-point, from which the disease spreads to other areas—the face, the neck, and the upper extremities are chiefly affected; the trunk is less often involved, and the legs are but seldom affected. In the earlier stages of the disease edematous spots appear; the skin in these areas becomes indurated and swollen, has a brawny, thick feel, and cannot be separated from the underlying tissues. Gradually the skin becomes more tense, and a disappearance of pigment

or abnormal pigmentation takes place. At this stage the skin presents a glossy, polished appearance. As the condition progresses the veins become prominent and the skin becomes thin, tense and cold, and is adherent to the subcutaneous tissue. The atrophy may extend to the deeper parts, and involve the muscles and bones. If the hands are involved, the fingers become stiff, thin, and shriveled; annular constriction may take place, with spontaneous amputation of the phalanges. When the face is involved, the movements of the facial muscles become restricted, the features are drawn, the face assumes a mask-like expression, the lips become thin and stretched, and mastication and speech are markedly interfered with.

If the neck and thorax are affected, respiration may be impeded. Sensibility is practically never affected. Mental depression commonly occurs, and in severe cases general wasting develops. Scleroderma may be combined with other trophic disorders, particularly facial hemiatrophy, Raynaud's disease, and erythromelalgia.

Course.—This disease usually begins gradually, being preceded by paresthesia. The affected areas are at first isolated, but gradually they become confluent. In a large majority of cases the condition is gradually progressive, lasting for many years.

Prognosis.—This disease usually terminates in death from cachexia or from some intercurrent disease.

Diagnosis.—As a rule, there is little difficulty in arriving at a correct diagnosis of this disease if one takes into consideration its gradual development and its characteristic features.

Treatment.—The general health of the patient should be carefully supervised. The bowels should be kept freely open and the diet regulated. General massage, galvanism of the spine, and inunctions of ichthyol ointment should be tried. Internally, the salicylates, the iodids, and thyroid and pituitary extract may be tried. Subcutaneous injections of a 15 per cent. alcoholic solution of thiosinamin, given every other day, are recommended by Hebra. Local treatment with hot air may also be tried.

UNCLASSIFIED DISORDERS

ACHONDROPLASIA

This condition is characterized by a congenital smallness of the stature, which is due to a deficient ossification of the cartilage of the long bones.

Etiology.—In some instances the disease is hereditary. The condition has been attributed to an intra-uterine inflammatory affection, during the course of which the cartilages at the epiphyseal ends of the long bones are destroyed, and, as a consequence, there is marked retardation in the longitudinal development of these bones.

Pathology.—This condition is the result of an interference in the growth of the cartilaginous portion of the skeleton. This interference with growth may begin at different periods of intra-uterine life—the earlier the condition begins, the shorter are the bones. Bones surrounded by membrane are not involved, the primary condition being a disturbance of cartilage formation.

Symptoms.—The average height of the achondroplastic subject varies from 38 to 53 inches. The shape of the hand is also characteristic: all the fingers are relatively of the same length; there is a peculiar separation of the second and the third finger at the second phalangeal joint which causes the digits to spread out, giving rise to the so-called "trident hand." Lumbar lordosis is present almost without exception. Cesarean section is practically always necessary when labor occurs in an achondroplastic woman (Fig. 169).

Even during the earliest years of life the small size and the shortness of the limbs are noticed. As the individual becomes older there is a marked retardation in the growth of the long bones, so that the individual remains of dwarfish stature. Cer-

tain peculiarities are characteristic of this disease. It is noticed that the head is of normal size, and is sometimes even larger than normal. The trunk also closely approaches the normal in

Fig. 169.—Achondroplasia, from Obstetrical Clinic, University of Maryland Hospital.

size, but is usually somewhat smaller. The most characteristic feature of the disease is the fact that the limbs, that is, the arms and legs, fail to develop in their long axes—the arms are very short when the individual is standing erect; the fingers usually

reach only to the trochanters, whereas in normal individuals they generally reach to the lower third of the thigh; the legs are also abnormally short. It is further noticed that the proximal ends of the limbs, that is, the arms and the thighs, are retarded in their longitudinal growth, even to a greater extent than are the distal ends. The musculature is well developed; the genitalia are normal in size and in function, and in the majority of cases the mentality is not affected.

Diagnosis.—This condition must be differentiated from rickets, cretinism, congenital syphilis, etc. From *rickets* the disease is differentiated by the fact that achondroplasia is congenital, the lesions being complete at birth; the deformities are present, but as the individual grows they become exaggerated; rickets, on the other hand, is a postnatal disease; furthermore, the *x*-ray aids in the differentiation of the two diseases: in achondroplasia the lesions are in the cartilage, and the epiphyses are about normal in size; in rickets the epiphyses are enlarged; in achondroplasia the head and trunk are normal, whereas in rickets they are frequently affected.

From *cretinism* this disease is differentiated by the fact that the mentality in achondroplasia is normal, whereas in cretinism there is a deficiency in intelligence; the hair in achondroplasia is abundant and soft, whereas in cretins it is scanty and coarse; the *x*-ray reveals the important difference in the bones; moreover, cretins improve under thyroid extract, which has no effect on the achondroplastic individual.

From *congenital syphilis* achondroplasia can be differentiated by *x*-ray examination of the bones.

Course.—This condition is present at birth, and the deformities are merely accentuated as the individual grows older. The prognosis as to life is good, but as to bringing about a normal growth of the stature it is absolutely hopeless.

Treatment.—Up to the present time no treatment is known to have any beneficial effect on this disease.

TETANUS

Etiology.—Tetanus is an acute infection caused by a bacillus that was discovered by Nicolaier. The organism enters the body through a wound of the skin or the mucous membranes. It is generally introduced deeply into the tissues, and grows enaĕrobically. As a result of the growth of this organism a toxemia is produced which causes the disease.

The spores of the bacillus may retain their virulence for many years. The bacillus or its spores are generally introduced into the body by some contaminating object, such as splinters, nails, glass, instruments, and the like. It may also be introduced as the result of administration of antitoxins, injections of gelatin, powder wounds, etc.

Tetanus occurs more frequently in warmer climates, owing to the fact that in these climates greater portions of the body are exposed because unprotected by clothing.

Pathology.—There is generally evidence of some local wound or abrasion, which may or may not be extensive. As the result of metabolic changes taking place during the growth of the tetanus bacillus introduced in the wound toxic substances are produced that at first cause local changes in the nerves adjacent to the injured parts. These are followed by changes in the cells in the anterior horns of the spinal cord and gray matter of the medulla.

Symptoms.—The first symptoms appear at variable periods after the injury, sometimes in three, four, or five days, and in some cases after several weeks.

Stiffness and contractions of the muscles of the face, body, and neck, which may be most mild or very marked, are usually the first symptoms to appear. Later these symptoms become more marked, spasm, and finally inability to open the mouth (trismus) occur. Soon after this opisthotonos occurs, and stiffness of the muscles of the larynx and pharynx, giving rise to difficulty in swallowing. The stiffness is followed by spasmodic contraction which increases from day to day, and rapidly spreads to involve the muscles of the trunk and extremities. The spasms spread from one group of muscles to another, following a

descending direction. The ocular muscles are rarely affected, and finally the respiratory muscles become involved, and breathing is carried on only with difficulty. The muscle contractions are accompanied by cramp-like pains.

Clonic spasmodic contractions occur in some cases. The intervals between the spasmodic attacks may be long or short. During these attacks the pain becomes much more marked. These attacks usually last only a few seconds, and are generally of reflex origin, being brought on by sudden noises, touching the patient, bright lights, odors, etc. Speech and deglutition are generally difficult and frequently impossible. The functions of the bowels and bladder may be impaired, and often there is retention of urine.

Sensation and the special senses are hardly ever affected. Consciousness is not affected and the mind generally remains clear. Profuse perspiration is an almost constant symptom. The quantity of urine excreted is usually decreased. The temperature is generally normal, but toward the end, in fatal cases, it rises and sometimes becomes very high. These individuals present a very characteristic appearance, generally lying in bed, breathing with difficulty, the features being rigidly contracted, the forehead furrowed, the eyelids drawn down, the mouth drawn to one side or the other, the jaws tightly closed, and the head drawn backward or to the side. The abdominal muscles are tense, the arms and the legs strongly extended, the muscles standing out prominently, and the facial expression showing that the patient is in pain. During sleep the muscles become relaxed.

In the type of tetanus known as head tetanus, or the hydrophobic tetanus of Rose, the condition is due to an infection of the face or skull with the tetanus bacillus. The symptoms of this type of infection usually occur eight or nine days after the infection has taken place. In this disease the first symptom to appear is spasm of the facial muscles on the same side as the injury; this is later followed by trismus, which is unilateral at first and later becomes bilateral. The muscular contractions spread to the pharynx and larynx and also affect the muscles of the neck. In a short time the facial spasm becomes converted

into a facial paralysis which is either partial or complete. In a few cases the facial paralysis is not preceded by a spasmodic condition. In some cases the muscular rigidity may extend still further and involve the muscles of the trunk and of the extremities.

Course.—The onset of the disease may be gradual and slowly progressive, or it may occur more or less suddenly and be rapidly progressive, beginning sometimes with evidences of local irritation in the region of the wound.

In mild cases the disease is limited to stiffness and contraction of the muscles of mastication and deglutition. In more marked cases the symptoms gradually progress until the muscles of the throat, trunk, and extremities are involved. Stiffness and contraction are the prominent symptoms.

In the severer cases the progress of the symptoms is rapid, and in addition to stiffness and contraction spasmodic attacks occur. The disease lasts from a few days to several weeks.

Prognosis.—In the majority of cases the prognosis is unfavorable, death usually occurring within five or six days; it may not occur, however, for several weeks, death being generally the result of asphyxia or exhaustion. In head tetanus death usually results from pneumonia, erysipelas, meningitis, exhaustion, etc.

The longer the interval between the infection and the appearance of the tetanic symptoms, the more favorable is the outlook. In those cases in which the disease takes a favorable course convulsive seizures and spasms finally become less marked and eventually the rigidity disappears. Trismus is the last symptom to disappear. Convalescence is usually protracted. If the symptoms are mild from the start and increase slowly, recovery may be expected. In those cases in which the first symptoms appear within five days after the injury and in puerperal tetanus the prognosis is generally very grave. In head tetanus the prognosis is usually more favorable than in general tetanus.

Diagnosis.—This disease must be differentiated from strychnin-poisoning, from hydrophobia, and from hysteric conditions. The differentiation from *strychnin-poisoning* is generally made from the history of the case and from the fact that generalized

convulsions are marked, whereas in tetanus the muscular contractions progress much more slowly, and the reflex convulsions are usually not so well marked. In tetanus the trismus is an early and prominent symptom; in strychnin-poisoning it is no more marked than any of the other muscle symptoms.

From *hydrophobia* the condition is differentiated from the history, the presence of permanent muscular rigidity, and the involvement of the jaw muscles first; whereas in hydrophobia the rigidity is not permanent, and the throat muscles are the first to be involved. From *hysteria* the differentiation is usually made with very little difficulty, as in this condition there is an absence of trismus and of permanent muscular rigidity, and the spasms, when they occur, are commonly purposive in character, and not indicative of a reflex origin.

Treatment.—The patient should be placed in a darkened, quiet room, and should be protected from all noise and excitement. He should be handled as little as possible, and every form of external irritation should be eliminated. He should be given an abundance of fluids and as nourishing a diet as possible; if necessary, the food may be given through a tube passed between the teeth or through the nose; in addition to this, nutritive enemata may be given.

The localized point of infection should be thoroughly cleansed and the wound enlarged and disinfected. Antitetanic serum should be freely injected into and about the seat of injury, and should also be given subcutaneously and intraspinously. In order that the serum may be effective it should be given early and in very large quantities. In order to prevent the development of tetanus it is most important that all wounds should receive proper surgical treatment, and those that are the result of punctures, occurring particularly on exposed surfaces of the body or in the presence of dirt, should, in addition to being treated surgically, have injected around and into them a moderately large amount of antitetanic serum.

Owing to the fact that the treatment of this condition with serum does not always result favorably, and also because of the fact that these patients generally suffer considerable discomfort, it is necessary, in addition to the administration of the serum, to

resort to other therapeutic measures. Chloral hydrate, given in doses of from 20 to 40 grains four, five, or six times a day, alone or combined with morphin, is one of the best remedies for relieving the muscle contractions. The patient should, so far as possible, be kept in a state of narcosis. Chloroform may also be used, especially to control the convulsions, and the bromids in large doses should always be given. The subcutaneous injection of about 30 minims of a 2 per cent. solution of phenol every two hours has also been recommended, and seems to be very effective in the treatment of this condition. Continuous warm baths may also be useful in relieving the muscle spasms. When the symptoms become less intense, it is very important that the patient's strength be sustained by a nourishing diet and an abundance of fluids. Care must be exercised to prevent the entrance of fluids or foods into the lungs. The condition of the bowels and bladder should also be carefully supervised.

INDEX

Abdominal neuralgia, 234
  reflex, 158
Abducens nerve, 129
    examination of, 173
Abscess of brain, 426
Accommodation, reaction to, 173
Achondroplasia, 641
Acromegaly, 605
Acroparesthesia, 184, 618
Acute ascending paralysis, 356
  circumscribed edema, 635
Agoraphobia, 513
Ala cinerea, 63
Alcohol, multiple neuritis due to, 213
Amaurotic family idiocy, 418
Amyotrophic lateral sclerosis, 298
Anemia, cerebral, 380
Angioneurotic edema, 635
Anterior poliomyelitis, acute, 278
    chronic, 286
    subacute, 286
Anterolateral ground bundle, function of, 55
Anxiety neurosis, 517
Apoplexy, 391
Apraxia, 165
Aqueduct of Sylvius, 62, 64
Arachnoid, 32
  pia, 21
Aran-Duchenne type of progressive atrophy, 286
Argyll Robertson's sign, 308
Arsenic, multiple neuritis due to, 216
Arterial system, central, 114
Arteries, posterior cerebral, 109
    communicating, 111
    inferior cerebellar, 109
    internal carotid, 109
  root, anterior, 50
    posterior, 50
  vertebral, 109
Artery, anterior cerebral, 110
    choroid, 113
    communicating, 110
    inferior cerebellar, 109
  basilar, 109
  middle cerebral, 111
  of cerebral hemorrhage, 111
  spinal anterior, 50
Ataxia, 313, 441
  hereditary, 298
  locomotor, 303
  Marie's cerebellar hereditary, 302
  static, 299
Ataxic paraplegia, 269
Athetoid movements, 149
Atrophic spinal paralysis, 278
Atrophy, optic, 254
  progressive, Aran-Duchenne type, 286
    spinal muscular, 286
Auditory nerve, 132
    disorders of, 263
Axon, 25

Babinski reflex, 158
Basedow's disease, 575
Basilar ganglia, 110, 117
Basis pedunculi, 65
Bell's paralysis, 259
Benedikt's syndrome, 269, 445
Bergeron's chorea, 544
Bergmann, 197
Beriberi, 220
Bernhard's disease, 201
Betz, cells of, 90
Biceps reflex, 154
Birth palsy of Duchenne, 194
Blepharoclonus, 524
Blepharospasm, 524
Blindness, hysteric, 481

Bonnet's sign, 236
Brachial neuralgia, 233
  neuritis, 194
  plexus, disease of, 180
Brain abscess, 426
  lesions, organic, with chorea, 547
  tumor, 440
Breast, neuralgia of, 234
Brissaud, 526
Broca's convolution, 85
Brown-Séquard syndrome, 334, 335
Bulbar paralysis, progressive, 274
Burdach, column of, 48

Caisson disease, 469
Calcarine fissure, 88
Caloric reaction, 175
Capsule, external, 108
  internal, functions of, 106
Caries, spinal, 319
Catalepsy, 414
Cauda equina, 32
Cells, functions of, 27
  of Betz, 90
  of Purkinje, 79
Central canal, 42
Cerebellar incision, anterior, 74
  tract, direct, 55
Cerebellum, 73
  functions of, 81
  microscopic structure of, 79
  peduncles of, 75
  structure of, 79
Cerebral anemia, 380
  arteries, occlusion of, 383
    rupture of, 383
  cortex, functions of, 116
  hyperemia, 382
  peduncles, 64, 90
  sinuses, thrombosis of, 403
  tumors, 433
  vein, internal, 99
Cerebrospinal fluid, 22, 146
    in tabes, 316
  meningitis, epidemic, 368
Cerebrum, 81
Cervicobrachial neuritis, 186
Cervico-occipital neuralgia, 233
Charcot's disease, 220
  joints, 316
Chiasm, optic, 92
Chippault's rule, 40, 338
Choked disk, 252
Chorda tympani, 131
Chorea accompanying organic brain lesions, 547
  Bergeron's, 544
  chronic progressive, 543
    hereditary, 543
  congenital, 547
  Dubini's, 549
  electric, 549
  epidemic, 548
  Henoch's, 550
  Huntington's, 543
  insaniens, 534
  minor, Sydenham's, 533
  paralytic, 538
Choreiform movements, 149
Chvostek's sign, 591
Circle of Willis, 90, 113
Circumflex nerves, paralysis of, 197
Claustrophobia, 513
Coal-tar products, neuritis due to, 219
Cochlear nerve, 133
Colitis, mucous, 504
Column, anterior, 45
  Burdach's, 48
  Goll's, 48
  lateral, 45
  posterior, 45
Commissure, anterior, 104
  gray, 104
  posterior, 42, 105
  white, 42
    posterior, 43
Congenital spinal malformation, 357
Conjunctival reflex, 160
Convolution, Broca's, 85
  occipitotemporal superior, 88
  paracentral, 88
Convulsions, 149
Corneal reflex, 160
Corpora quadrigemina, 65
    functions of, 71
  striata, 100
Corpus callosum, 102
Corrugator supercilii reflex, 156
Cortex, cerebral, functions of, 116
Cranial nerves, anatomy, 123
    diseases of, 250
    physiology, 123

Cremasteric reflex, 158
Cretinism, 583
Crises, gastric, 305
  intestinal, 307
  laryngeal, 307
  pharyngeal, 307
  rectal, 307
Crura, disease of, 269
  functions of, 71
Crural neuralgia, 234
Crusta, 70
Cuneus, 88

Deep sensibility, 55, 163
Degeneration, process of, 181
  reaction of, 170, 183
  Wallerian law of, 27
Dementia, paralytic, 449
Dendrons, 24
Dentate fascia, 33
Diadokokinesis, 151
Dipsomania, 515
Direct cerebellar tract, function of, 55
Disk, choked, 252
Dislocation of spinal column, 325
Disseminated sclerosis, 465
Double personality, 476
Dubini's chorea, 549
Duchenne, birth palsy of, 194
  pseudomuscular hypertrophy of, 240
  subacute ascending paralysis, 286
Duchenne-Landouzy-Déjérine type of dystrophy, 240
Dura mater, 19
Dysbasia lordotica, 530
Dystrophy, Duchenne-Landouzy-Déjérine type, 240
  Erb's juvenile, 240
  progressive muscular, 239

Edema, acute circumscribed, 635
  angioneurotic, 635
Electric chorea, 549
  examination, 167
Encephalitis, acute non-suppurative hemorrhagic, 408
Endoneurium, 31
Epicritic sensibility, 55, 162
Epidemic chorea, 548
Epilepsy, 557
  Jacksonian, 558
  myoclonus, of Unverricht, 572
  nocturnal, 558
  psychic, 557
Epineurium, 31
Equilibrium, 123
Erb's combined shoulder-arm paralysis, 190
  juvenile dystrophy, 240
  sign, 592
Erythromelalgia, 627
Examination, electric, 167
  nerve, abducens, 173
    facial, 174
    glossopharyngeal, 175
    hypoglossal, 176
    motor oculi, 173
    optic, 172
    patheticus, 173
    pneumogastric, 175
    spinal accessory, 175
    trifacial, 174
  of nervous system, 147
  of patient, 142
  physical, 145
Exhaustion, nervous, 493
Exophthalmic goiter, 575
Exophthalmos, 577
External cutaneous nerve, neuritis of, 201
  popliteal nerve, neuritis of, 204

Facial hemiatrophy, progressive, 637
  nerve, diseases of, 259
    examination of, 174
  spasm, 522
  tic, 524
Falling sickness, 557
Family idiocy, amaurotic, 418
Fasciæ dentatæ, 33
Fasciculus cuneatus, 61
  gracilis, 61
  of Rolando, 61
Fever, hysteric, 483
Fibers, external arcuate, 60
  of Remak, 27
Fifth nerve disease, 258
Fillet, 69

Fissure, anterior median, 35
anterolateral, 58
calcarine, 88
callosomarginal, 87
corpus callosum, 87
dentate, 88
hippocampal, 88
of Rolando, 82
of Sylvius, 82
parieto-occipital, 82, 88
posterior median, 35
subparietalis, 87
Focal symptoms, 440
Foerster's method, 421
operation, 317
Folie du doute, 514
Foot-drop, 213
Foramen of Magendie, 35, 60
of Monro, 96
of Retzius, 35, 60
Formatio reticularis, 67
Fornix, 105
Fourth nerve, paralysis of, 256
Fracture of spinal column, 325
Frenkel's operation, 317
Fröhlich's syndrome, 599

Gait, scissors, 418
steppage, 215, 220
Galvanic current, 169
Ganglia, basal, 100, 117
Ganglion, geniculate, 131
Ganglionic system, central, 114
Gangrene, symmetric, 629
General paralysis of insane, 449
paresis, 449
Geniculate body, external, 66
internal, 66
ganglion, 131
Gersuny's paraffin injection, 639
Gigantism, 603
Gliosis, spinal, 292
Globus hystericus, 474
Glossolabiopharyngeal paralysis, 274
Glossopharyngeal nerve, 133
disease of, 264
examination of, 175
Goiter, exophthalmic, 575
Goll, column of, 48
functions of, 55
Grand mal, 557, 561
Graves' disease, 575
Groove, posterolateral, 59
Ground-bundle, anterolateral, 46, 48
function of, 55

Habit spasms, 530
Hand, trident, 640
Hematomyelacia, 349
Hemianopsia, unilateral nasal, 251
Hemiatrophy, progressive facial, 637
Hemicrania, 620
Hemiplegia, 393
Hemorrhage, cerebral, artery of, 111
meningeal, 361
Henoch's chorea, 550
Hereditary ataxia, 298
syphilis, 461
Herpes zoster, 222
Hippocampus major, 96
Hoffman's sign, 591
Horn, lateral, 42
posterior, 42
Huntington's chorea, 543
Hydrocephalus, 422
Hydrophobia, 647
Hyperemia, cerebral, 382
Hypertrophic pachymeningitis, 341
Hypertrophy, pseudomuscular, of Duchenne, 240
Hypoglossal nerve, 58, 136
examination of, 175
Hypophysis cerebri, 92
functions of, 117, 598
Hypotonia, 309
Hysteria, 471
major, 474
minor, 474
Hysteric fever, 483
ptosis, 481
vomiting, 483
Hysterogenic zones, 165, 480

Idiocy, amaurotic family, 418
Illuminating gas, multiple neuritis due to, 219
Imperative idea, 513
Infantile spinal paralysis, 287
Infantilism, 611
cachectic type, 612
ductless gland, 613

Infantilism, Lorain type, 611
Insane, general paralysis of, 449
Intention tremor, 466
Intercostal nerves, paralysis of, 200
  neuralgia, 233
Internal popliteal nerve, neuritis of, 204
Interstitial neuritis, 181
Ischemic paralysis, 246
Island of Reil, 84
Isthmus, 88

Jacksonian epilepsy, 558
Jaw reflex, 155
Jonnesco's operation, 583

Kleptomania, 515
Klumpke's paralysis, 191
Knee reflex, 153
Korsakoff's syndrome, 214

Lancisi, nerves of, 103
Landry's paralysis, 356
Lateral sclerosis, 295
Lead-poisoning, multiple neuritis due to, 219
Lemniscus, 69
Leprous neuritis, 222
Light sensibility, 162
Lissauer's marginal tract, 46, 48
Little's disease, 417
Lobe, central, 84, 87
  frontal, 84
  limbic, 84, 88
  occipital, 85
  olfactory, 84, 85
  parietal, 85
  temporal, 86
Lobule, quadrate, 88
Locomotor ataxia, 303
Locus cœruleus, 62
Lorain type of infantilism, 611
Lumbar neuralgia, 234
  plexus, disease of, 201

Magendie, foramen of, 35, 60
Marie's cerebellar hereditary ataxia, 302
Martin's sign, 236
Mastication, spasm of muscles of, 526
Mastodynia, 234
Medulla, disease of, 269
  function of, 71
  structure of, 66
Medullary velum, anterior, 62
    superior, 77
Meningeal hemorrhage, 361
Meningismus, 378
Meningitis, 364
  chronic, 343
  epidemic cerebrospinal, 368
  secondary, 365
  septic, 365
  serous, 378
  spinal, 339
  syphilitic, 375
  tubercular, 372
Meralgia paræsthetica, 201
Migraine, 620
Möbius' sign, 579
Monro, foramen of, 96
Morton's toe, 205
Motor impulse, course of, 117
  nerve, 30, 31
  oculi nerve, 173
  system, examination of, 148
Movements, athetoid, 149
  choreiform, 149
Mucous colitis, 504
Multiple neuritis, 206
    course, 210
    diagnosis, 211
    due to alcohol, 213
      to arsenic, 216
      to coal-tar products, 219
      to illuminating gas, 219
      to lead-poisoning, 219
    etiology, 207
    prognosis, 210
    symptoms, 207
    treatment, 210
  sclerosis, 465
Muscle sense, 56
  spasms, localized, 522
Muscular dystrophy, progressive, 239
  system, disease of, 239
Musculospiral nerve, paralysis of, 197
Myasthenia gravis, 277, 594
Myasthenic reaction, 170, 595
Myatonia congenita, 247

Myelitis, 344
Myoclonus epilepsy of Unverricht, 572
multiplex, 573
Myotonia congenita, 247
Myotonic reaction, 170
Mysophobia, 513
Myxedema, 583

Nerve, abducens, 129
examination of, 173
auditory, 132
circumflex, paralysis, 197
cochlear, 133
facial, 130
diseases of, 259
examination of, 174
fifth, disease of, 258
fourth, paralysis of, 256
glossopharyngeal, 133
disease of, 264
hypoglossal, 58, 136
motor, 30, 31
musculospiral, paralysis of, 197
obturator, disease of, 201
oculomotor, 125
olfactory, 123
optic, 125
disorders of, 251
examination of, 172
phrenic, paralysis of, 186
pneumogastric, 61, 135
sciatic, 203
sensory, 30, 31
sixth, paralysis of, 256
spinal accessory, 61, 127
suprascapular, paralysis of, 197
trifacial, 127
tumors of, 225
ulnar, paralysis of, 199
Nerve-fiber, medullated, 27
non-medullated, 27
structure of, 36
Nerve-root, anterior, 37
posterior, 37
Nerves, cranial, 123
diseases of, 250
of Lancisi, 103
Nervous exhaustion, 493
system, examination of, 147
sympathetic, 136
Nervous system, sympathetic, functions of, 139
syphilis of, 459
Neuralgia, 226
abdominal, 234
brachial, 233
cervico-occipital, 233
course, 228
crural, 234
diagnosis, 228
etiology, 227
intercostal, 233
lumbar, 234
of breast, 234
pathology, 226
prognosis, 228
sciatic, 235
symptoms, 227
treatment, 229
trigeminal, 230
Neurasthenia, 493
Neuritis, brachial, 194
cervicobrachial, 186
course, 185
etiology, 182
external cutaneous, 201
internal popliteal, 204
interstitial, 181
leprous, 222
multiple, 206. See also *Multiple neuritis*.
of sacral plexus, 201
optic, 253
parenchymatous, 181
plantar, 205
popliteal, external, 204
postdiphtheritic, 219
prognosis, 175
puerperal, 220
symptoms, 182
treatment, 186
Neurogliar tissue, 42, 513
Neuron, 23
Neurosis, anxiety, 517
occupation, 518
Nocturnal epilepsy, 558
Node of Ranvier, 26
Nucleus cuneatus, 62
gracilis, 62
red, 70
trigeminal, 62
Nyctophobia, 513

Obsessions, intellectual, 514
volitional, 514
Obstetric paralysis (mother), 194, 202
Obturator nerve, disease of, 201
Occipital sinus, 20
Occupation neurosis, 518
pareses, 206
spasms, 518
Oculomotor nerve, 125
Olfactory nerve, 123
disease of, 250
Olives, 59
Ophthalmoplegia, 257
external, 257
internal, 257
Oppenheim's disease, 247
Optic atrophy, 254
chiasm, 92
nerve, 125
disorders of, 251
examination of, 172
neuritis, 253
tracts, 92

Pachymeningitis, external, 355
hypertrophic, 341
internal, 359
Pain, 162
sense, 121
Palsy, shaking, 550
Paræsthesia, 163
Paraffin injection, Gersuny's, 639
Paralysis, acute ascending, 356
agitans, 550
atrophic spinal, 278
Bell's, 259
Erb's combined shoulder-arm, 190
general, of insane, 449
glossolabiopharyngeal, 274
infantile spinal, 287
ischemic, 246
Klumpke's, 191
Landry's, 356
obstetric, 194, 202
of circumflex, nerves, 197
of intercostal nerves, 200
of musculospiral nerve, 197
of phrenic nerve, 186
periodic, 249
progressive bulbar, 274
sixth nerve, 256
Paralysis, subacute ascending, of Duchenne, 286
ulnar nerve, 199
Volkmann's, 246
Paralytic chorea, 538
dementia, 449
Paramyoclonus multiplex, 573
Paraplegia, ataxic, 269
senile, 355
Parathyroid glands, 590
Parenchymatous neuritis, 181
Paresis, general, 449
occupation, 206
Peduncles, cerebral, 64, 90
cerebelli inferior, 77
middle, 77
superior, 76
Perineum, 31
Periodic paralysis, 249
Periosteoradial reflex, 154
Personality, double, 476
Petit mal, 557, 562
Pharyngeal reflex, 160
Phrenic nerve, paralysis of, 186
Physical examination, 145
Pineal gland, 102
disorders of, 615
Pituitary body, 92
disorders of, 597
function of, 117, 598
symptoms due to disturbance of secretion, 599
Plantar nerves, neuritis of, 205
Plexus, arterial, anterior, 50
Pneumogastric nerve, 61, 135
examination of, 175
Points of Valleix, 235
Poliomyelitis, acute anterior, 278
chronic, 286
subacute, 286
Polymyositis, 244
Pons, functions of, 71
structure of, 66
varolii, 62
Postdiphtheritic neuritis, 219
Postepileptic mental states, 564
Posterior inferior cerebral artery, occlusion of, 270
perforated space, 92
Præcuneus, 88
Progressive spinal muscular atrophy, 286

Projection tract, 115
Protopathic sensibility, 163
Pseudomuscular hypertrophy of Duchenne, 240
Pseudotabes alcoholica, 215
Psychalgia, 228
Psychic epilepsy, 557
Ptosis, hysteric, 481
Puerperal neuritis, 220
Puncture, spinal, 176
Purkinje, cells of, 79
Putamen, 101
Pyramidal tract, crossed, 46, 47
direct, 46, 47

Quadrate lobule, 88

Ranvier, node of, 26
Raynaud's disease, 629
Reaction, caloric, 175
myasthenic, 170, 595
myotonic, 170
of degeneration, 170, 183
tetanic, 170
to accommodation, 173
Recklinghausen's disease, 225
Rectal reflex, 161
Reflex, abdominal, 158
Babinski, 158
biceps, 154
conjunctival, 160
corneal, 160
corrugator supercilii, 156
cremasteric, 158
jaw, 155
knee, 153
periosteoradial, 154
pharyngeal, 160
rectal, 161
superficial, 157
tendo Achillis, 152
triceps, 155
vesical, 161
Reflexes, 152
Regeneration, 28
Reid's line, 179
Reil, island of, 84
Remak, fibers of, 27
Rest-cure, Weir-Mitchell's, 510
Retzius, foramina of, 35, 60
Rolando, fasciculus of, 61
fissure of, 82
Romberg's sign, 150, 309
Roots, anterior, 43
posterior, 43
Rubrospinal tract, 71
Rump's sign, 501

Sacral plexus, neuritis of, 201
Schwann, sheath of, 26
Sciatic nerve, 203
neuralgia, 235
Sciatica, 235
Scissors gait, 418
Scleroderma, 639
Sclerosis, amyotrophic lateral, 295, 298
disseminated, 465
multiple, 465
Secretory disturbance, 165
Senile paraplegia, 355
Sensation, 55
deep, 55, 163
Sense, muscle, 56
pain, 121
stereognostic, 165
Sensibility, protopathic, 163
Sensory function, 162
impulse, course of, 117
nerve, 30, 31
Shaking palsy, 550
Sheath of Schwann, 26
Sign, Bonnet's, 236
Chvostek's, 591
Erb's, 592
Hoffman's, 591
Martin's, 236
Möbius, 579
Romberg's, 150, 309
Rump's, 501
Stellwag's, 579
von Graefe's, 579
Westphal's, 308
Sinus, longitudinal, 20
occipital, 20
straight, 20
Sinuses, lateral, 20
Sixth nerve paralysis, 256
Somnambulistic attacks, 476
Space, subarachnoid, 21, 33
Spasm, 149

Spasm, facial, 522
Spasmodic torticollis, 526
Spasms, habit, 530
  localized muscle, 522
  occupation, 518
  of muscles of mastication, 526
Spina bifida, 357
Spinal accessory nerve, 61, 127
    disease of, 267
    examination of, 175
  canal, syphilitic exudation into, 325
  caries, 319
  column, fracture and dislocation of, 325
  cord, anatomy of, 32
    blood supply of, 50
    cellular constituents of, 53
    physiology of, 32, 53
    pressure on, 319
    segment of, 38
      functions, 57
    structure of, 40
    tumors of, 331
  gliosis, 292
  malformation, congenital, 357
  meningitis, 339
  puncture, 176
Spinothalamic tract, 69
Static ataxia, 299
Status epilepticus, 563
Stellwag's sign, 579
Steppage gait, 215, 220
Stereognostic sense, 165
St. Vitus' dance, 533
Subarachnoid space, 21, 33
Substantia nigra, 70
Suprascapular nerve, paralysis of, 197
Sydenham's chorea, 533
Sylvius, aqueduct of, 62, 64
  fissure of, 82
Symmetric gangrene, 629
Sympathetic nervous system, 136
    functions of, 136
Syndrome, Benedikt's, 269, 445
  Brown-Séquard's, 334
  Fröhlich's, 599
  Korsakoff's, 214
  Weber's, 269
Syphilis, hereditary, 461
  of nervous system, 459
Syphilitic exudation into spinal canal, 325
Syringomyelia, 292

Tabes dorsalis, 303
    cerebrospinal fluid in, 316
Tabetic arthropathies, 311
Tachycardia, 576
Tactile sense, 121, 162
Tangential tract, 114
Tegmentum, 65, 70
Tela choroidea, inferior, 60
Temperature sense, 121, 162
Tenderness of nerve-trunks on pressure, 163
Tendo Achillis reflex, 152
Tentorium cerebelli, 73
Tetanic reaction, 170
Tetanus, 644
  head type, 645
  hydrophobic, 645
Tetany, 589
Thomsen's disease, 247
Thrombosis, infective, 405
  of cerebral sinuses, 403
  of lateral sinus, 406
  of longitudinal sinus, 405
Tic, 530
  douloureux, 231
  facial, 524
Tinnitus aurium, 264
Toe, Morton's, 205
Torticollis, spasmodic, 526
Tortipelvis, 530
Tract, anterolateral, 69
  cerebellar direct, 46, 47
  comma, 46
  comma-shaped, Schultze's, 50
  commissural, 114
  Gower's, 46, 47
  marginal, Lissauer's, 46, 48
  of cord, functions of, 54
  optic, 92
  projection, 115
  pyramidal, crossed, 46, 47
    direct, 46, 47
  rubrospinal, 71
  spinothalamic, 69
  tangential, 114
  ventrospinocerebellar, 69
Tremor, intention, 466

Tremors, 148, 557
Triceps reflex, 155
Trident hand, 641
Trifacial nerve, 127
examination of, 174
Trigeminal neuralgia, 230
Trigonum hypoglossi, 62
Trophic disturbances, 166
Trophoneuroses, 637
Tuber cinereum, 92
Tumors, cerebral, 433
nerve, 127
of spinal cord, 331

Ulnar nerve, paralysis of, 199
Unverricht, myoclonus epilepsy of, 572

Valleix, points of, 235
Valve of Vieussens, 64, 77
Vasomotor disturbances, 165
Velum, medullary, anterior, 62
superior, 77
Ventricle, fourth, 60, 62
third, 96
Ventricles, lateral, 93
Ventrospinocerebellar tract, 69
Vertebræ, malignant growths of, 323
Vertebral arteries, 109
Vesical reflex, 161
Vieussens, valve of, 64, 77
Volkmann's paralysis, 246
Vomiting, hysteric, 483
Von Graefe's sign, 579

Wallerian law of degeneration, 27
Weber's syndrome, 269
Weir-Mitchell's rest cure, 510
Westphal's sign, 308
Willis, circle of, 90, 113
Wrist-drop, 213

Zones, hysterogenic, 165, 480

SAUNDERS' BOOKS

on

Practice, Pharmacy, Materia Medica, Therapeutics, Pharmacology, and the Allied Sciences

W. B. SAUNDERS COMPANY

West Washington Square — **Philadelphia**

9, Henrietta Street — **Covent Garden, London**

Our Handsome Complete Catalogue will be Sent You on Request

Medical Clinics of Chicago

Issued serially, one octavo of 200 pages, illustrated, every other month. Per Clinic Year (July to May), six numbers: Cloth, $12.00 net.

EXCLUSIVELY INTERNAL MEDICINE

These bi-monthly publications are devoted exclusively to *Clinical Internal Medicine* in all its departments. They give you the bedside and amphitheater teachings of leading Chicago internists, representing such large hospitals as Mercy, Cook County, St. Luke's, Michael Reese, and Sarah Morris Memorial. The widest variety of cases is included, bringing out forcibly every feature of history-taking, diagnosis, treatment, and general management. The cases are illustrated with *x*-ray pictures, photographs, pulse-tracings, and temperature charts; the technic of all laboratory tests is given in detail, and every aid that can serve to make the diagnosis and treatment of the cases thoroughly clear to the general practitioner is emphasized. These publications are *clinical in the strictest sense*—they are an exposition of diagnosis and treatment as *actually practiced* at the bedside and in the amphitheater.

Musser and Kelly on Treatment

A Handbook of Practical Treatment. By 82 eminent specialists. Edited by JOHN H. MUSSER, M. D., and A. O. J. KELLY, M. D., University of Pennsylvania. Three octavo volumes, averaging 950 pages each, illustrated. Per volume: Cloth, $6.00 net; Half Morocco, $7.50 net.

THE TREATMENT THAT IS ALL TREATMENT

Every chapter in this work was written by a specialist of unquestioned authority. Not only is drug therapy given, but also dietotherapy, serumtherapy, organotherapy, rest-cure, exercise and massage, hydrotherapy, climatology, electrotherapy, *x*-ray, and radial activity are fully, clearly, and definitely discussed. Those measures partaking of a *surgical nature* have been presented by *surgeons*.

The Medical Record

"The most modern and advanced views are presented. It is difficult to pick out any one topic that deserves special commendation, all parts fully covering their particular field, and written with that fulness of detail demanded by the every-day needs of the practitioner."

Thomson's Clinical Medicine

Clinical Medicine. By WILLIAM HANNA THOMSON, M.D., LL. D., formerly Professor of the Practice of Medicine and of Diseases of the Nervous System, New York University Medical College. Octavo of 667 pages. Cloth, $5.00 net; Half Morocco, $6.50 net.

A RECORD OF 50 YEARS

This new work represents over *a half century of active practice and teaching*. It deals with *bedside* medicine—the *application* of medical knowledge for the relief of the sick. First the meaning of common and important symptoms is stated *definitely;* then follows a chapter on the use of remedies and a classification of them; next the section on infections, and last a section on diseases of particular organs and tissues. An important chapter is that on the mechanism of surface chill and "catching cold," going very clearly into the etiologic factors, and outlining the treatment. The chapter on remedies takes up non-medicinal and medicinal remedies and *vaccine and serum therapy.* The infectious diseases are taken up in Part II, while Part III deals with diseases of special organs or tissues, every disease being fully presented from the *clinical side.*

Tousey's Medical Electricity, Röntgen Rays, and Radium

Medical Electricity, Röntgen Rays, and Radium. By SINCLAIR TOUSEY, M. D., Consulting Surgeon to St. Bartholomew's Hospital, New York. Octavo of 1219 pages, with 801 illustrations, 19 in colors. Cloth, $7.50 net; Half Morocco, $9.00 net.

NEW (2d) EDITION, RESET

The revision for this edition was extremely heavy; new matter has increased the size of the book by some 100 pages. About 50 new illustrations have been added. The new matter added includes: Diathermy, sinusoidal currents, radiography with intensifying screens, röntgenotherapy, the Coolidge and similar Röntgen tubes and the author's method of dosage, and radium therapy. The book has been enriched by including several of Machado's tabular classifications of electric methods, effects, and uses.

Throughout the entire work everything concerning electricity, *x*-rays, and radium in medicine, as well as phototherapy, is explained in detail—nothing is omitted. It tells you how to equip your office, and, more than that, how to use your apparatus, explaining away all difficulties. It tells you just how to apply these measures in the treatment of disease. The chapters on *dental radiography* are particularly valuable to those interested in dental work.

Abbott's Medical Electricity

MEDICAL ELECTRICITY. By GEORGE KNAPP ABBOTT, M. D., Dean and Professor of Physiologic Therapy and Practice, College of Medical Evangelists, Loma Linda, California. 12mo of 132 pages, illustrated. Cloth, $1.25 net.

This new work gives the nurse the essentials of this subject. Dr. Abbott's style has made the difficult simple. The text is illustrated.

Gant's Work on Diarrhea

Diarrhea, Inflammatory and Parasitic Diseases of the Gastro-intestinal Tract. By SAMUEL G. GANT, M. D., LL.D., Professor of Diseases of the Sigmoid Flexure, Colon, Rectum, and Anus, New York Post-Graduate Medical School and Hospital. Octavo of 604 pages, with 181 illustrations. Cloth, $6.00 net; Half Morocco, $7.50 net.

ILLUSTRATED

This new work is particularly full on the two practical phases of the subject —*diagnosis* and *treatment*. For instance: While the essential diagnostic points are given under each disease, a fuller description of diagnostic methods is given in a special chapter. The *differential diagnosis* of diarrheas of local and those of systematic disturbances is strongly brought out. There is a special chapter on *nervous diarrheas* and those originating from *gastrogenic* and *enterogenic dyspepsias*. You get the psychotherapy of psychic diarrheas. You get reliable methods of simultaneously controlling associated constipation and diarrhea. You get a complete *formulary*—prescriptions from Dr. Gant's own practice. There is a chapter on hookworms, tapeworms and round worms, and on the diarrheas caused by them and other parasites. This chapter contains many *excellent illustrations*. The limitations of drugs are pointed out, the dangers of their use emphasized, and the indications for surgical intervention given. You get the *technic in detail* of all surgical procedures indicated—fully illustrated.

Gant's Intestinal Stasis (Constipation and Obstruction)

This work is medical, non-medical (mechanical), and surgical, the latter really being a complete work on rectocolonic surgery. The chapters on *therapeutic gymnastics* and *massage* are the outgrowth of Dr. Gant's personal experience. You get practical articles on *diverticulitis*, *peridiverticulitis*, *pericolitis*, *perisigmoiditis* (*Jackson's membrane*), *Lane's kink*, and affections of the *ileocecal valve*.

Octavo of 575 pages, with 250 illustrations. By SAMUEL G. GANT, M. D., LL. D., New York Post-Graduate Medical School and Hospital. Cloth, $6.00 net; Half Morocco, $7.50 net.

"The best and most complete treatise on these subjects."—*Medical Record.*

Cabot's Differential Diagnosis

Differential Diagnosis. Presented through an analysis of 385 Cases. By RICHARD C. CABOT, M.D., Assistant Professor of Clinical Medicine, Harvard Medical School. Two octavos of 750 pages each, illustrated. Per volume: Cloth, $5.50 net; Half Morocco, $7.00 net.

VOLUME 1 [New (3d) Edition—Just Out]: Headache, pain in various regions, fevers, chills, coma, convulsions, weakness, cough, vomiting, hematuria, dyspnea, jaundice, and nervousness—21 symptoms and 385 cases.

VOLUME 2 (Just Out): Abdominal and other tumors, vertigo, diarrhea, dyspepsia, hematemesis, enlarged glands, blood in stools, swelling of face, hemoptysis, edema of legs, frequent micturition and polyuria, fainting, hoarseness, pallor, swelling of arm, delirium, palpitation and arhythmia, tremor, ascites and abdominal enlargement—19 symptoms and 317 cases.

Morrow's Diagnostic and Therapeutic Technic

Diagnostic and Therapeutic Technic. By ALBERT S. MORROW, M.D., Adjunct Professor of Surgery, New York Polyclinic. Octavo of 830 pages, with 860 original line drawings. Cloth, $5.00 net.

NEW (2d) EDITION

Dr. Morrow's new edition is decidedly a work for you—the physician engaged in general practice. It is a work you need because it tells you just how to perform those procedures required of you every day, and it tells you and *shows* you by clear, *new* line-drawings, in a way never before approached. The information it gives is such as you need to know every day—transfusion and infusion, hypodermic medication, Bier's hyperemia, exploratory punctures, aspirations, anesthesia, etc.

Journal American Medical Association

"The procedures described are those which practitioners may at some time be called on to perform."

Garrison's History of Medicine

History of Medicine. With Medical Chronology, Bibliographic Data, and Test Questions. By FIELDING H. GARRISON, M. D., Principal Assistant Librarian, Surgeon-General's Office, Washington, D. C. Octavo of 763 pages, illustrated. Cloth, $6.00 net; Half Morocco, $7.50 net.

THE BAEDEKER OF MEDICAL HISTORY

The work begins with ancient and primitive medicine, and carries you in a most interesting and instructive way on through Egyptian medicine, Sumerian and Oriental medicine, Greek medicine, the Byzantine period; the Mohammedan and Jewish periods, the Medieval period, the period of the Renaissance, the Revival of learning and the Reformation; the Seventeenth Century (the age of individual scientific endeavor), the Eighteenth Century (the age of theories and systems), the Nineteenth Century (the beginning of organized advancement of science), the Twentieth Century (the beginning of organized preventive medicine). You get all the important facts in medical history; a *biographic dictionary* of the makers of medical history, arranged alphabetically; an album of *medical portraits;* a complete *medical chronology* (data on diseases, drugs, operations, etc.); a brief survey of the *social and cultural phases* of each period.

McKenzie on Exercise

Second Edition

Exercise in Education and Medicine. By R. TAIT MCKENZIE, B. A., M. D., Professor of Physical Education, and Director of the Department, University of Pennsylvania. Octavo of 585 pages, with 478 illustrations. Cloth, $4.00 net.

D. A. Sargent, M. D., *Director of Hemenway Gymnasium, Harvard University.*

"It cannot fail to be helpful to practitioners in medicine. The classification of athletic games and exercises in tabular form for different ages, sexes, and occupations is the work of an expert. It should be in the hands of every physical educator and medical practitioner."

Carter's Diet Lists

DIET LISTS OF THE PRESBYTERIAN HOSPITAL OF NEW YORK CITY. Compiled, with notes, by HERBERT S. CARTER, M. D. 12mo of 129 pages. Cloth, $1.00 net.

Bonney's Tuberculosis

Second Edition

TUBERCULOSIS. By SHERMAN G. BONNEY, M. D., Professor of Medicine, Denver and Gross College of Medicine. Octavo of 955 pages, with 243 illustrations. Cloth, $7.00 net; Half Morocco, $8.50 net.

Anders' Practice of Medicine

A Text-Book of the Practice of Medicine. By JAMES M. ANDERS, M. D., PH. D., LL. D., Professor of the Practice of Medicine and of Clinical Medicine, Medico-Chirurgical College, Philadelphia. Handsome octavo, 1332 pages, fully illustrated. Cloth, $5.50 net; Half Morocco, $7.00 net.

NEW (12th) EDITION

The success of this work is no doubt due to the extensive consideration given to Diagnosis and Treatment, under Differential Diagnosis the points of distinction of simulating diseases being presented in tabular form. In this new edition Dr. Anders has included all the most important advances in medicine, keeping the book within bounds by a judicious elimination of obsolete matter. A great many articles have also been rewritten.

Wm. E. Quine, M. D., *College of Physicians and Surgeons, Chicago.*

"I consider Anders' Practice one of the best single-volume works before the profession at this time, and one of the best text-books for medical students."

DaCosta's Physical Diagnosis

Physical Diagnosis. By JOHN C. DACOSTA, JR., Associate Professor of Medicine, Jefferson Medical College. Octavo of 589 pages, with original illustrations. Cloth, $3.50 net.

NEW (3d) EDITION

In Dr. DaCosta's work every method given has been carefully tested and proved of value by the author himself. Normal physical signs are explained in detail in order to aid the diagnostician in determining the abnormal. Both direct and differential diagnoses are emphasized. The 243 original illustrations are artistic as well as practical.

Henry L. Elsner, M. D., *Professor of Medicine, Syracuse University.*

"I have reviewed this book and am thoroughly convinced that it is one of the best ever written on the subject. In every way I find it a superior production."

Sahli's Diagnostic Methods

Edited by Nath'l Bowditch Potter, M.D.

A Treatise on Diagnostic Methods of Examination. By Prof. Dr. H. Sahli, of Bern. Edited, with additions, by Nath'l Bowditch Potter, M.D., Assistant Professor of Clinical Medicine, Columbia University. Octavo of 1225 pages, profusely illustrated. Cloth, $6.50 net.

SECOND EDITION, RESET

Lewellys F. Barker, M. D.
Professor of Medicine, Johns Hopkins University

"I am delighted with it, and it will be a pleasure to recommend it to our students in the Johns Hopkins Medical School."

Friedenwald & Ruhräh on Diet

Diet in Health and Disease. By Julius Friedenwald, M. D., Professor of Diseases of the Stomach, and John Ruhrah, M. D., Professor of Diseases of Children, College of Physicians and Surgeons, Baltimore. Octavo of 857 pages. Cloth, $4.00 net.

NEW (4th) EDITION

Dietetic management in all diseases in which diet plays a part in treatment is carefully considered, the articles on diet in diseases of the digestive organs containing numerous diet lists and explicit instructions for administration. The feeding of infants and children, of patients before and after anesthesia and surgical operations, are all taken up in detail.

"It seems to me that you have prepared the most valuable work of the kind now available. I am especially glad to see the long list of analyses of different kinds of food."—George Dock, M. D., *Tulane University of Louisiana.*

Eggleston's Prescription Writing

This new work is a crystallization of Dr. Eggleston's long experience in teaching this subject. It covers the entire field in a most practical way, taking up grammar, construction, dosage, vehicles, incompatibility, administration, etc.

16mo of 115 pages. By Cary Eggleston, M. D., Instructor in Pharmacology at Cornell University Medical School. Cloth $1.00 net.

Kemp on Stomach, Intestines, and Pancreas

Diseases of the Stomach, Intestines, and Pancreas. By ROBERT COLEMAN KEMP, M. D., Professor of Gastro-intestinal Diseases at the New York School of Clinical Medicine. Octavo of 1025 pages, with 377 illustrations. Cloth, $6.50 net; Half Morocco, $8.00 net.

NEW (2d) EDITION

It is the practitioner who first meets with these cases, and it is he upon whom the burden of diagnosis rests. After the diagnosis is established, the practitioner, if properly equipped, could frequently treat the case himself instead of transferring it to a specialist. This work is intended to equip the practitioner with this end in view.

The Therapeutic Gazette

"The therapeutic advice which is given is excellent. Methods of physical and chemical examination are adequately and correctly described."

Bastedo's Materia Medica, Pharmacology, Therapeutics, and Prescription Writing

By W. A. BASTEDO, M. D., Associate in Pharmacology and Therapeutics at Columbia University. Octavo of 602 pages, illustrated. Cloth, $3.50 net.

THREE PRINTINGS IN SIX MONTHS

Dr. Bastedo's new work has the distinct advantage of presenting the subjects from both the laboratory and the clinical sides. Dr. Bastedo for many years devoted his entire time to laboratory work. Now, however, he is strictly a *clinical* man. He gives you the practical, daily application of that information he gleaned at first hand in the laboratory—facts you can use in your bedside practice. Because of this early laboratory training you are assured that his book is *correct* according to *laboratory standards*. Being now a strictly clinical man, you know that his book is modeled with the common purpose of all medical practice: *The treatment of the sick.*

Faught's Blood-Pressure

Blood-Pressure from the Clinical Standpoint. By FRANCIS A. FAUGHT, M. D., formerly Director of the Laboratory of Clinical Medicine of the Medico-Chirurgical College of Philadelphia. Octavo of 475 pages, illustrated.

NEW (2d) EDITION

Dr. Faught's book is designed for practical help *at the bedside.* It meets the urgent needs of the general practitioner, who heretofore had no book to which to turn in case of emergency. Every effort has been made to provide here a practical guide, full of information of a clinical nature, and presented in a way readily available for daily use by the busy man. Besides the actual technic of using the sphygmomanometer in diagnosing disease, Dr. Faught has included a brief general discussion of the process of circulation. The practical application of sphygmomanometric findings within recent years makes it imperative for every medical man to have close at hand an up-to-date work on this subject.

Anders and Boston's Medical Diagnosis

A Text-Book of Medical Diagnosis. By JAMES M. ANDERS, M.D., PH.D., LL. D., Professor of the Theory and Practice of Medicine and of Clinical Medicine, and L. NAPOLEON BOSTON, M. D., Professor of Physical Diagnosis, Medico-Chirurgical College, Philadelphia. Octavo of 1248 pages, with 466 illustrations. Cloth, $6.00 net.

NEW (2d) EDITION

This new work is designed expressly for the general practitioner. The methods given are practical and especially adapted for quick reference. The diagnostic methods are presented in a forceful, definite way by men who have had wide experience at the bedside and in the clinical laboratory.

The Medical Record

"The association in its authorship of a celebrated clinician and a well-known laboratory worker is most fortunate. It must long occupy a pre-eminent position."

Deaderick & Thompson's Endemic Diseases of South

ENDEMIC DISEASES OF THE SOUTHERN STATES. By WILLIAM H. DEADERICK, M. D., Member American Society of Tropical Medicine; and LOYD THOMPSON, M. D., Charter Member American Association of Immunologists. Octavo of 546 pages, illustrated. Cloth, $5.00 net; Half Morocco, $6.50 net.

This new work is really a collection of monographs on malaria, blackwater fever, pellagra, amebic dysentery, hookworm disease, and other intestinal parasites. Diagnosis, prophylaxis, and treatment are gone into in detail, giving you every aid to the correct interpretation of the symptoms presented, and every modern means of value in the prevention and treatment of the diseases discussed.

Smith's What to Eat and Why **Second Edition**

WHAT TO EAT AND WHY. By G. CARROLL SMITH, M. D., Boston. 12mo of 377 pages. Cloth, $2.50 net.

With this book you no longer need send your patients to a specialist to be dieted—you will be able to prescribe the suitable diet yourself, just as you do other forms of therapy. Dr. Smith gives "the why" of each statement he makes. It is this knowing why which gives you confidence in the book, which makes you feel that Dr. Smith *knows*.

Ward's Bedside Hematology

BEDSIDE HEMATOLOGY. By GORDON R. WARD, M. D., Fellow of the Royal Society of Medicine, London, England. Octavo of 394 pages, illustrated. Cloth, $3.50 net.

Slade's Physical Examination & Diagnostic Anatomy

PHYSICAL EXAMINATION AND DIAGNOSTIC ANATOMY. By CHARLES B. SLADE, M. D., Chief of Clinic in General Medicine, University and Bellevue Hospital Medical College. 12mo of 146 pages, illustrated. Cloth, $1.25 net.

Stevens' Therapeutics

Fifth Edition

A TEXT-BOOK OF MODERN MATERIA MEDICA AND THERAPEUTICS. By A. A. STEVENS, A.M., M.D., Lecturer on Physical Diagnosis in the University of Pennsylvania. Octavo of 675 pages. Cloth, $3.50 net.

Dr Stevens' Therapeutics is one of the most successful works on the subject ever published. In this new edition the work has undergone a very thorough revision, and now represents the very latest advances.

The Medical Record, New York

"Among the numerous treatises on this most important branch of medical practice, this by Dr. Stevens has ranked with the best."

Butler's Materia Medica

Sixth Edition

A TEXT-BOOK OF MATERIA MEDICA, THERAPEUTICS, AND PHARMACOLOGY. By GEORGE F. BUTLER, PH.G., M.D., Professor and Head of the Department of Therapeutics and Professor of Preventive and Clinical Medicine, Chicago College of Medicine and Surgery, Medical Department Valparaiso University. Octavo of 702 pages, illustrated. Cloth, $4.00 net; Half Morocco, $5.50 net.

For this sixth edition Dr. Butler has entirely remodeled his work, a great part having been rewritten. All obsolete matter has been eliminated, and special attention has been given to the toxicologic and therapeutic effects of the newer compounds.

Medical Record, New York

"Nothing has been omitted by the author which, in his judgment, would add to the completeness of the text."

Sollmann's Pharmacology

Second Edition

A TEXT-BOOK OF PHARMACOLOGY. By TORALD SOLLMANN, M.D., Professor of Pharmacology and Materia Medica, Western Reserve University. Octavo of 1070 pages, illustrated. Cloth, $4.00 net.

The author bases the study of therapeutics on systematic knowledge of the nature and properties of drugs, and thus brings out forcibly the intimate relation between pharmacology and practical medicine.

J. F. Fotheringham, M.D., *Trinity Medical College, Toronto.*

"The work certainly occupies ground not covered in so concise, useful, and scientific a manner by any other text I have read on the subjects embraced."

Arny's Pharmacy

PRINCIPLES OF PHARMACY. By HENRY V. ARNY, PH. G., PH. D., Professor of Pharmacy, New York College of Pharmacy. Octavo of 1175 pages, with 246 illustrations. Cloth, $5.00 net.

George Reimann, Ph. G., *Secretary of the New York State Board of Pharmacy.*

"I would say that the book is certainly a great help to the student, and I think it ought to be in the hands of every person who is contemplating the study of pharmacy."

Hinsdale's Hydrotherapy

Hydrotherapy: A Treatise on Hydrotherapy in General; Its Application to Special Affections; the Technic or Processes Employed, and the Use of Waters Internally. By GUY HINSDALE, M. D., Fellow of the Royal Society of Medicine of Great Britain. Octavo of 466 pages, illustrated. Cloth, $3.50 net.

The Medical Record

"We cannot conceive of a work more useful to the general practitioner than this, nor one to which he would resort more frequently for reference and guidance in his daily work."

Kelly's Cyclopedia of American Medical Biography

Cyclopedia of American Medical Biography. By HOWARD A. KELLY, M. D., Johns Hopkins University. Two octavos of 525 pages each, with portraits. Per set: Cloth, $10.00 net; Half Morocco, $13.00 net.

Dr. Kelly, in these two handsome volumes, presents concise, yet complete biographies of those men and women who have contributed noteworthily to the advancement of medicine in America. Dr. Kelly's reputation for painstaking care assures accuracy of statement. There are about one thousand biographies included.

Swan's Prescription-writing and Formulary

PRESCRIPTION-WRITING AND FORMULARY. By JOHN M. SWAN, M.D., Director Glen Springs Sanitarium, Watkins, N. Y. 12mo of 185 pages. Flexible cloth, $1.25 net.

Stewart's Pocket Therapeutics and Dose-book

New (4th) Edition

POCKET THERAPEUTICS AND DOSE-BOOK. By MORSE STEWART, JR., M.D. 32mo of 263 pages. Cloth, $1.00 net.

Bohm and Painter's Massage

MASSAGE. By MAX BOHM, M. D., of Berlin, Germany. Edited, with an Introduction, by CHARLES F. PAINTER, M. D., Professor of Orthopedic Surgery at Tufts College Medical School, Boston. Octavo of 91 pages, with 97 *practical* illustrations. Cloth, $1.75 net.

GET THE BEST

American Illustrated Dictionary

THE NEW STANDARD

The New (8th) Edition, Reset

The American Illustrated Medical Dictionary. By W. A. NEWMAN DORLAND, M. D., Editor of "The American Pocket Medical Dictionary." Octavo of 1137 pages. Flexible leather, $4.50 net; with thumb index, $5.00 net.

OVER 1500 NEW WORDS

Howard A. Kelly, M. D., *Johns Hopkins University, Baltimore.*

"Dr. Dorland's dictionary is admirable. It is so well gotten up and of such convenient size. No errors have been found in my use of it."

Thornton's Dose-Book

Fourth Edition

DOSE-BOOK AND MANUAL OF PRESCRIPTION-WRITING. By E. Q. THORNTON, M. D., Assistant Professor of Materia Medica, Jefferson Medical College, Philadelphia. Post-octavo, 392 pages, illustrated. Flexible leather, $2.00 net.

"It will afford me much pleasure to recommend the book to my classes, who often fail to find such information in their other text-books."—C. H. MILLER, M.D *Professor of Pharmacology, Northwestern University Medical School, Chicago.*

Lusk on Nutrition

Second Edition

ELEMENTS OF THE SCIENCE OF NUTRITION. By GRAHAM LUSK, PH.D., Professor of Physiology in Cornell University Medical School. Octavo of 402 pages. Cloth, $3.00 net.

"I shall recommend it highly. It is a comfort to have such a discussion of the subject."—LEWELLYS F. BARKER, M. D., *Professor of the Principles and Practice of Medicine, Johns Hopkins University.*

Hatcher and Sollmann's Materia Medica

A TEXT-BOOK OF MATERIA MEDICA: including Laboratory Exercises in the Histologic and Chemic Examination of Drugs. By ROBERT A. HATCHER, PH. G., M. D.; and TORALD SOLLMANN, M. D. 12mo of 411 pages. Flexible leather, $2.00 net.

Bridge on Tuberculosis

TUBERCULOSIS. By NORMAN BRIDGE, A. M., M. D. 12mo of 302 pages, illustrated. Cloth, $1.50 net.

American Pocket Dictionary — New (9th) Edition

THE AMERICAN POCKET MEDICAL DICTIONARY. Edited by W. A. NEWMAN DORLAND, M.D. Flexible leather, with gold edges, $1.00 net; with thumb index, $1.25 net.

Pusey and Caldwell on X-Rays — Second Edition

THE PRACTICAL APPLICATION OF THE RONTGEN RAYS IN THERAPEUTICS AND DIAGNOSIS. By WILLIAM ALLEN PUSEY, A. M., M. D., and EUGENE W. CALDWELL, B. S. Octavo of 625 pages, with 200 illustrations. Cloth, $5.00 net.

Cohen and Eshner's Diagnosis. Second Revised Edition

ESSENTIALS OF DIAGNOSIS. By S. SOLIS-COHEN, M. D., and A. A. ESHNER, M. D. Post-octavo, 382 pages; 55 illustrations. Cloth, $1.00 net. *In Saunders' Question-Compend Series.*

Morris' Materia Medica and Therapeutics — Seventh Edition

ESSENTIALS OF MATERIA MEDICA, THERAPEUTICS, AND PRESCRIPTION-WRITING. By HENRY MORRIS, M. D. Revised by W. A. BASTEDO, M. D., Instructor in Materia Medica and Pharmacology, Columbia University. 12mo, 300 pages. Cloth, $1.00 net. *Saunders' Compends.*

Deaderick on Malaria

PRACTICAL STUDY OF MALARIA. By WILLIAM H. DEADERICK, M. D., Member American Society of Tropical Medicine. Octavo of 402 pages, illustrated. Cloth, $4.50 net.

Goepp's State Board Questions — Third Edition

STATE BOARD QUESTIONS AND ANSWERS. By R. MAX GOEPP, M. D., Professor of Clinical Medicine, Philadelphia Polyclinic. Octavo of 715 pages. Cloth, $4.00 net.

Niles on Pellagra — New (2d) Edition

PELLAGRA. By GEORGE M. NILES, M. D., Gastro-enterologist to the Georgia Baptist Hospital, Atlanta. Octavo of 253 pages, illustrated. Cloth, $3.00 net.

Arnold's Medical Diet Charts

MEDICAL DIET CHARTS. Prepared by H. D. ARNOLD, M. D., Professor of Clinical Medicine, Tufts Medical College, Boston. Single charts, 5 cents; 50 charts, $2.00 net; 500 charts, $18.00 net; 1000 charts, $30.00 net.

Saunders' Pocket Formulary

Ninth Edition

SAUNDERS' POCKET MEDICAL FORMULARY. By WILLIAM M. POWELL, M. D. Containing 1900 formulas from the best-known authorities. In flexible leather, with side index, wallet, and flap. $1.75 net.

Jakob and Eshner's Internal Medicine and Diagnosis

ATLAS AND EPITOME OF INTERNAL MEDICINE AND CLINICAL DIAGNOSIS. By DR. CHR. JAKOB, of Erlangen. Edited, with additions, by A. A. ESHNER, M. D. 182 colored figures on 68 plates, 64 text-cuts, 259 pages of text. Cloth, $3.00 net. *In Saunders' Hand-Atlas Series.*

Lockwood's Practice of Medicine

Second Edition Revised and Enlarged

A MANUAL OF THE PRACTICE OF MEDICINE. By GEO. ROE LOCKWOOD, M. D., Attending Physician to the Bellevue Hospital, New York City. Octavo, 847 pages, illustrated. Cloth, $4.00 net.

Fenwick's Dyspepsia

DYSPEPSIA. By WILLIAM SOLTAU FENWICK, M. D., of London. Octavo of 485 pages, illustrated. Cloth, $3.00 net.

Jelliffe's Pharmacognosy

AN INTRODUCTION TO PHARMACOGNOSY. By SMITH ELY JELLIFFE, PH. D., M. D., Columbia University, New York. Octavo of 265 pages, illustrated. Cloth, $2.50 net.

Stevens' Practice of Medicine

New (10th) Edition

A MANUAL OF THE PRACTICE OF MEDICINE. By A. A. STEVENS, A. M., M. D., Professor of Therapeutics and Clinical Medicine, Woman's Medical College, Philadelphia. 12mo, 573 pages, illustrated. Flexible leather, $2.50 net.

Camac's Epoch-Making Contributions

EPOCH-MAKING CONTRIBUTIONS TO MEDICINE AND SURGERY. By C. N. B. CAMAC, M. D., of New York City. Octavo of 450 pages, with portraits. Artistically bound, $4.00 net.

Todd's Clinical Diagnosis

New (3d) Edition

CLINICAL DIAGNOSIS. By JAMES CAMPBELL TODD, M. D., Professor of Pathology, University of Colorado, Denver. 12mo of 585 pages, illustrated. Cloth, $2.50 net.

COLUMBIA UNIVERSITY LIBRARIES

This book is due on the date indicated below, or at the expiration of a definite period after the date of borrowing, as provided by the rules of the Library or by special arrangement with the Librarian in charge.

DATE BORROWED	DATE DUE	DATE BORROWED	DATE DUE
	FEB 20 1948		
FEB 20 1948			
C28(1141)M100			

RC 341
S53
Shear
Manual of Nervous Diseases